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LECTURES ON

CLINICAL MEDICINE,

DELIVERED AT THE HÔTEL-DIEU, PARIS.

BY

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VOLUME FIFTH.

WITH INDEX.

TRANSLATED FROM THE EDITION OF 1868,

Being the Third Revised and Enlarged Edition;

BY

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Gentlemen:—In 1858, we received into Saint-Agnes's ward three persons with intermittent fever: one of them contracted this disease in the Crimea, and the other two in Africa. They returned to France, and came to reside in Paris, in 1857; but it was not till 1858, six months afterwards, that these individuals suffered from a recurrence of paroxysms of intermittent fever for which they had been treated, and apparently with complete success, in the country in which they were first attacked.

We frequently see recurrence of the paroxysms of marsh fever in persons for a long time removed from the influences which first produced the symptoms. This recurrence, common though it be, is not the less deserving of the attention of physicians: it is not without importance in relation to nosology, but its relations to therapeutics are still more important.

We must be careful not to confound these recurrences, more or less distant in respect of time, and more or less possessing the character of periodicity, with morbid phenomena proceeding from a single persistent cause, with a diathesis of the organism, or with relapses of pyrexiæ and phlegmasiæ.

A person, for example, after the lapse of weeks, months, or years, takes, a second time, a purely inflammatory peripneumonia: this second attack does not consist of new manifestations of an existing disease, but is on each occasion a new disease, exactly similar to the first in its seat and nature, like it, too, running through all the stages, and accomplishing, *uno tenore*, its complete evolution. If we admit the existence in the individual of a peculiar predisposition to pulmonary inflammation, we must suppose that in each attack, there is a new intervention of the same cause, or of some analogous cause, the effects of which would entirely exhaust themselves in a series of uninterrupted morbid acts, till there either is a return to previous health, or a fatal issue.

It is not so, however, in respect of diathesic diseases, with which marsh fever has so much analogy, that I feel some difficulty in not placing it in the same class.

At some particular epoch, an indiviuual is attacked by gout: some months later, he has a second and then a third seizure: each of these seizures is not a new disease, but only a new manifestation of the same disease of which the cause, never completely exhausted, though remaining quiet for a longer or shorter period, has not the less existed in power within the economy—existed *in posse* to make use of an old medical expression. Though under certain circumstances, an immediate cause becomes the starting point of the accidents, the intervention of that cause is not absolutely necessary, as in peripneumonia of which I have just now been speaking.

A similar remark is applicable to pox. Whatever time may have elapsed between the different outbreaks of characteristic symptoms, these symptoms are always subordinate to one and the same cause.

A similar statement applies to marsh fever.

It certainly never would occur to any one, that the periodic paroxysms of a quotidian, tertian, or quartan intermittent constitute so many distinct illnesses. It is palpable to every one, that it is always the same disease, the manifestations of which are separated, by longer or shorter intervals, during which intervals, the morbific cause remaining latent, the economy has seemed to regain its perfect equilibrium of health. It is not, however, so generally believed, or at all events is not so generally stated, that the periods of respite may be exceedingly prolonged. I am not alluding to intermittent fevers said to have monthly or annual paroxysms, cases of which have been collected by excellent authors, among whom may be mentioned Schenck: I refer only to cases similar to those presented by our three patients in Saint-Agnes's ward.

You observe that in them, after an interval of six months, during which the individuals remained quite well, the manifestations of intermittent fever which they had had on the first occasion, and of which they had been apparently completely cured, have not been reproduced without an appreciable determining cause. In them, it has been the same fever which has made a new attack; and this has occurred, as on the first occasion, under the influence of an infection, the germ of which had not been wholly destroyed.

The germ of marsh fever not only sometimes remains quiescent for months or years after its first manifestation; but it also happens that individuals who have received the germ in countries where marsh fevers are endemic, do not experience the first apparent symptoms till long afterwards, when perhaps living in countries where such fevers do not generally prevail. Cases of this kind are by no means rare; and two have lately presented themselves to your notice, almost simultaneously, at this hospital. In one case, the patient was a child admitted in July to Saint-Bernard's ward with intermittent fever, evidently contracted in Burgundy where he had been reared, and whence he was brought six months ago to Paris. In the other case, the patient was a man whom some of you must have seen in the wards of my friend and colleague, Dr. Delpech. A certain time has elapsed since this man returned from Africa. Enlarged spleen and a straw-yellow colour of skin indicated that he was under the influence of serious paludal cachexia; and he came to the hospital to be treated for intermittent fever, which he assured us had first declared itself a few days previously, not the slightest paroxysm of fever ever having occurred in Algeria, where he had received the germ of the disease.

Gentlemen, from these remarks, you will already have seen the analogy which I pointed out between marsh fevers and diathesic diseases: in them, as in every diathesis, you will find that there is a morbific cause which may remain quiescent, for a longer or shorter period, in the organism which it affects. The analogy will present itself in a still more striking light when I explain to you the diversity of the manifestations of this morbific cause.

Bear in mind my definition of a diathesis. It is a special state, a peculiar disposition of the economy, hereditary or *acquired*, but essentially and invariably chronic, in virtue of which are produced disturbances in general pretty intense, which are identical in their nature, but variable and fluctuating in their form.

These disturbances of the system, variable and fluctuating in form, resting on the same basis, depending on one and the same cause, are met with in the disease now under our consideration. Though this disease manifests itself generally by intermittent and periodic attacks of fever, though its periodic character is so generally present that the term *intermittent* coupled with the designation of the fever is sufficient to define the nosological species, still, it frequently happens, that it assumes other features: the diathesis, and the paludal poison clothe it in forms essentially different from those in which it is generally clothed. It behoves the physician to be able to recognise the disease under this diversity of form, so as to be able to combat it.

The term *paludal* sufficiently indicates the nature of the diathesis, which in fact it describes. It recognises that its ordinary cause is sojourn in places poisoned by paludal miasmata. In passing, let me mention, as a remarkable circumstance, that the marsh miasm seems to be a poison only to the human species. Few human beings can expose themselves to it with impunity, yet the lower animals, speaking at least from what we observe in respect of domestic animals, are not at all affected by it.

Though paludal affections are nowhere more common than in countries which are hot, low, and damp, you must not conclude that they are engendered by humidity of climate. Humidity only acts when there is considerable extent of surface covered with water, or when the soil is impregnated with water, which evaporates under the influence of certain atmospheric conditions, this evaporation producing telluric emanations, which perhaps constitute what we call the "marsh miasmata."

To so small an extent is humidity the cause of intermittent fevers, that one may without much inconvenience reside in a marshy district during the cold season, and during the rainy season, so long as the ground is submerged. But whenever the ground dries up, or even when it becomes partially exposed by the action of the heat of the day, there is danger. Whether it be a marsh properly so called, the bed of a river or rivulet, or an inundated meadow, marsh miasmata are developed, which poison those exposed to their emanations.

Again, even in dry and parched regions, similar results follow great disturbance of soil which has not been stirred for a very long period. Hence it is that in highly cultivated districts—on the elevated *plateau* of the Beauce, for example—where there is a constant scarcity of water, very many cases of intermittent fever are met with.

The paludal affections which so cruelly ravaged our army during the war in the East, had no other cause than the great upturning of the soil necessitated by the siege works before Sebastopol.

Even here, in Paris, where intermittent fevers are of such rare occurrence that we hardly know them, they have shown themselves on several similar occasions. In 1811, during the digging of the Saint-Martin canal, a veritable epidemic of intermittent fever prevailed in the districts of the Temple, the Villette, and Pantin. In 1840, there was a similar epidemic when the fortifications which now surround the capital were being raised. The diggings of later years required in piercing the town by new streets and boulevards, the construction of sewers and underground passages for the gas pipes, have caused numerous paludal affections; and more than one physician has been surprised by meeting with cases of pernicious fever which he was little accustomed to encounter in his practice.

It must be stated that these paludal maladies, occurring accidentally as it were in countries which are usually exempt from them, are generally of a much less serious character than those which prevail as permanent endemics in localities where unfavourable conditions of humidity and temperature coexist. It is in such localities that we observe all the different manifestations of the diathesis, ranging from the intermittent fevers, which constitute the simplest expression of the diathesis, up to cases representing the very highest degree of the pathogenic action of poisonous miasmata.

The prejudicial influence of these insalubrious localities sometimes extends to a considerable distance beyond them, in consequence of atmospheric currents frequently transporting the marsh effluvia to places considerably remote.

The following fact, mentioned by Lancisi, and repeated in your text books, is one with which all of you are acquainted. Thirty inhabitants of Rome were walking towards the mouth of the Tiber: all at once, the wind arose, blowing across the marshes and carrying with it their emanations: twenty-nine of the thirty were seized with intermittent fever. Similar examples of the remote diffusion of paludal miasmata are not uncommon in the French districts of

Sologne, Bourbon, and Bresse, as well as in other permanent centres of intermittent fever. If it be sufficient in many cases for a stranger to traverse a marshy country to take marsh fever, how much more liable to do so must be those who habitually reside in them: the diathesis impresses its stamp, or if I may use such an expression, becomes much more deeply rooted in the inhabitants, who, besides the miasmata, have other deplorable hygienical conditions to contend against.

The paludal diathesis does not always manifest itself by fevers: in very many residents in districts where marsh fevers are endemic, it announces its existence by organic changes, more or less characterised, and by an assemblage of morbid phenomena obvious to the observer.

There is a loss of colour in the integuments, corresponding to a particular alteration in the blood: it is not merely the paleness perceived in persons exhausted by hemorrhages, nor is it the green hue of the chlorotic: it is a more or less deep tint of yellowish bistre.

This special cahectic tint, when strongly marked, generally coexists with engorgement of the spleen and liver, particularly of the spleen, which becomes much enlarged, and can easily be measured by percussion and palpation. Its increased bulk may be so great as to cause a decided projection through the abdominal parietes; and the enlarged organ may fill, as it were, the whole abdominal cavity, extending from the right beyond the median line, descending into the left iliac fossa, ascending into the cavity of the thorax, pushing up the diaphragm so as to embarrass the breathing, and, by exerting pressure on the vessels, assisting to produce those serous effusions, which I told you are frequent complications of this deplorable condition of the economy.

It is not unusual, as I observed in Sologne, for infants to be born with these visceral engorgements and the cachectic tint of the skin, testifying that even in the womb they had been under the same evil influence as that in which their mothers had lived.

Generally, however, before reaching this state of profound cachexia the individuals have experienced acute symptoms of paludal poisoning, acute phenomena of fever, which, according to the intensity of its cause, assume either the intermittent type, which we are accustomed to consider as the truly characteristic expression of miasmatic poisoning, or the remittent or continued type: this was clearly pointed out by the older physicians, particularly by Lancisi and Morton; and to our accomplished brethren of the army, Boudin, Laverau, and Maillot, we are indebted for having recalled attention to the fact.[1]

It is when these marsh fevers are of old date—even when the patients have left the countries in which they were first attacked—that the consequent anæmia and cachexia increase, becoming more and more marked. Then also supervene the serous effusions of which I have been speaking; œdema of the extremities, general anasarca, peritoneal and pleural dropsy, œdema of the lungs—affections which depend upon an alteration of the blood, and which (ascites and œdema of the lower extremities at least) have, to a certain extent, as their determining cause, the impediment to the portal circulation and to the passage of the blood through the vena cava produced by the enlarged liver and spleen.

When I come to speak of pernicious fevers, it will be seen that the spleen is sometimes the seat of very serious symptoms, of hemorrhages similar to those which occur within the encephalon, of softenings more or less extensive, and of more or less considerable ruptures which bring speedily mortal maladies in their train.

Every writer on intermittent fever has called attention to this cachexia and its accompanying engorgements of the abdominal viscera. Van Swieten, in noticing what his predecessors have said on this subject, adds, that in some cases, the dilatation and gorging of the colon, in its portions adjoining the spleen, are so great as to lead one to believe that the organ is tumefied.

You know, Gentlemen, the part which has recently been assigned to the hypertrophied spleen: some physicians, as you are aware, resting upon the notion put forth by Dr. Audouard, and developed by him in different memoirs, hold that congestion of the spleen is the cause of the periodicity of the paroxysms.[2] This theory, which does not maintain its ground when the facts are seriously examined, has very few defenders in the present day, and consequently I shall not stop to discuss it. Let me only remark to you that this is no new idea, Galen having said, in several places in his works, that the spleen is the headquarters of intermittent fever.

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[1] BOUDIN:—Traité des Fièvres Intermittentes, Rémittentes, et Continues. Paris: 1842.

MAILLOT:—Traité des Fièvres ou Irritations Cérébro-Spinales Intermittentes. Paris: 1838.

[2] AUDOUARD:—Des Congestions Sanguines de la Rate. Paris. 1848.

This remark brings me to the consideration of the great question of the *cause of intermittence,* a question often discussed, but never yet solved.

Some years ago, the following explanation was proposed by Dr. Masurel, a military physician. He said that the beginning of an intermittent fever is a vitiation of the blood by poisonous miasmata: by the ganglionic nervous system receiving an impression from the altered blood comes the paroxysm of fever, or rather the shivering, which is simply a symptom indicating the existence of a true neuropathic condition, a depressing heteronervation of the circulatory system: the second and third stages of the paroxysm, and the hot and sweating stages are results of the reaction of the organism from the profound disturbance occasioned by the two conditions which presided at the development of the paroxysm. When the paroxysm is past, there still remain miasmatic principles in the blood, which, by their ceaseless round in the circulation, induce a new change in the nervous system; whence come a new paroxysm and a new circle of fever and remission.

This theory, which seems to derive its inspiration from Cullen's views regarding the cause of fever, has a great similarity to the opinion of Sydenham, who believed that the paroxysm of intermittent fever returns because the morbific matter, after accumulating in the system during the apyrexial period and being eliminated by the sweating, reaccumulates, till a renewed effort of nature again solicits its elimination.

To these statements, Van Swieten made the following peremptory reply:—Although evacuations bring on crises in acute diseases, imperfect evacuations induce only imperfect crises, which never occur within such a period of time and at such regular intervals as in intermittent fevers.

Again, were the recurrence of the paroxysm dependent upon a remaining excess of morbific matter not expelled in the previous crisis, it would still remain to be discovered, why the potency of this morbific matter increases with greater or less rapidity according to the different kinds of fever—why, for example, it acts only after twelve hours in the quotidian, and only after thirty-six hours in fevers of the tertian type. How does it come to pass that an individual struck by a quartan should feel perfectly well a quarter of an hour before the return of the paroxysm, although the morbific matter then brought into action, must have been previously quite ready to act? How can we admit that in one and the same variety of intermittent fever affecting individuals differing in age, sex, and temperament, the morbific matter should take exactly the same time to accumulate and produce its effects? So inconsiderable are the evacuations essential to these fevers that, when they are treated by cinchona, recovery takes place without sweating, purging, or any unusual flow of urine, although it has been maintained by some persons that a cure obtained without critical evacuation is neither radical nor certain.

Finally, Gentlemen, although everything leads to the belief that the cause of intermittence is in the nervous system, we are not acquainted with that cause. However, this much at least we know, that intermittence pertains essentially to the organism, and not to the action on it of an external cause, although we see it occur in physiological order without appreciable causes, and in the pathological order under the influence of the most diverse causes. Some of these causes, however, are more susceptible than others of exciting it, even of soliciting it with a constancy and regularity which is very often periodic.

The poison of marsh miasm is one of these causes. Though under certain circumstances, to which I have already alluded, it manifests its action by phenomena invested with the continued type, intermittence usually characterises the phenomena induced by this morbific cause. The accidents of this description which are most common, or at least which seem to be best known to the majority of physicians, are those which constitute REGULAR INTERMITTENT FEVERS.

In exceptional cases, these fevers have precursory symptoms, such as general discomfort, accompanied by a feeling of debility, by lassitude and headache: the patient has pandiculation, and eructations. Generally, the attack begins abruptly; its paroxysms being composed of three distinct periods, called *stages*—the *cold*, *hot*, and *sweating* stages.

The *cold stage*, which ought rather to be called the *shivering stage*, because the sensation of cold experienced by the patient is merely the result of perverted sensibility, the temperature in reality changing but little, and even sometimes rising several degrees in certain parts of the body, a fact established by the experiments of Haen, which have been repeated by others. The cold stage is characterised by a shivering fit of more or less violence, of variable duration, but always rather long.

The shivering, which seems to begin in the lumbar region and ascend along the vertebral column, is soon trembling and real convulsive movement, affecting, in the first instance, the maxillary muscles, and so strongly as to cause the teeth to strike one another with a chattering noise. The trunk and limbs are then, and almost simultaneously, seized in such a manner that the rapid shocks by which the whole body is agitated shake the bed on which the patient is lying. The dryness of the skin, with its projecting papillæ giving it that appearance called *goose's skin*: the decoloration of the skin, nowhere more evident than in the face and extremities, which sometimes assume a slightly bluish tint, testifies to the disturbance which has supervened in the capillary circulation; while, at the same time, the smallness, feebleness, frequency, and irregularity of the pulse show that there is embarrassment of the arterial circulation.

There is great anxiety, marked oppression, intense thirst, the tongue remaining moist and sometimes bluish: there is complete loss of appetite: and this state of extreme discomfort is not unfrequently increased by fits of vomiting. The urine is scanty, pale, and aqueous.

These phenomena continue for one or two hours, and in some rare cases for three or four hours; but never longer, unless when the fever is an algid intermittent, which does not belong to the legitimate intermittents of which alone I am now speaking, but is one of the pernicious fevers which we shall afterwards have to consider.

The *hot stage* commences by the rigors becoming more and more transient, and alternating with warm flushings. The heat, slight at first, grows increasingly intense, and becomes sharp, biting, and very painful. The patients vary their position in bed, seeking a somewhat cooler place. The degree of heat is far from being in relation to the preceding shivering; and is often much less in reality than it would appear to be from the statements of the patients. Modern researches have shown that the temperature of the body, as ascertained by the thermometer, is, in many cases, not more than one degree above that which it indicated during the cold stage. However, to the touch, the skin is dry and hot: the face is red and animated—the eyes brilliant, and sometimes very sensitive to light.

The pulse loses its hardness, without diminishing in frequency: it gains in amplitude and power. The respiration is less anxious, deeper, and more frequent. The headache, with which the attack began, increases in place of diminishing; and in some cases, there is a little delirium. The anorexia and thirst continue. The urine has a more or less deep colour. The average duration of this stage is from one to two hours. In some patients, the skin becomes moist as soon as the shivering ceases.

In due course, these symptoms moderate; and the *sweating stage* sometimes announces itself by a general feeling of comparative well-being. The skin relaxes, little by little, becomes moist, and is soon covered by a profuse sweat. In some subjects, vomiting and slight diarrhœa seem to indicate that a similar emunction is taking place from the internal integument. The urine, red when it is passed, deposits on cooling a sediment of the color of brick-dust. This sediment, however, which some have looked upon as an essential phenomenon in all attacks of intermittent fever, is not always found: the urine may be thick or yellowish white resembling mare's urine, or slightly cloudy, or finally, it may differ very little from normal urine.

The cessation of the fever is announced by a diminution in the frequency of the pulse, which preserves its fulness.

Generally, as soon as calm is restored to the system, the patient falls asleep: this restorative sleep sometimes comes along with the sweating.

So terminates the paroxysm of a legitimate intermittent fever: it is succeeded by a period of repose, the *apyrexial period,* which, according to the kind of fever, lasts for a longer or shorter time.

When the apyrexia is of long duration, the patient entirely forgets, so to speak, the paroxysm which he has traversed, and fancies that he is restored to perfect health; but when the period of repose is short, or when the paroxysms for a time recur frequently, the repose is never absolute. There may then be observed general discomfort, an indescribable feeling of fatigue, slight heaviness of the head, a certain degree of anorexia coincident with a saburral state of the tongue, thirst, and irregularity in the alvine evacuations, showing that the functions have not entirely regained their normal action.

Turgescence of the liver and spleen, particularly of the latter, in persons who have been for a considerable period affected with these fevers, is a very significant phenomenon.

The apyrexial period, according to its duration, is characteristic of the different *types* which intermittent fever may assume. Gentlemen, you know what these types are.

The *quotidian* fever nas its paroxysms nearly the same, returning daily almost at the same hour. Let me here call your attention to a very important fact. In intermittent marsh fevers, the paroxysm generally commences in the morning, or at least before noon: on the other hand, the attacks of certain *symptomatic* intermittent fevers, also quotidian, such as those so often accompanying tubercular disease and the commencement of some pyrexiæ, supervene towards evening.

The *tertian* consists of paroxysms returning every two days, that is to say, on the third day, counting that on which the first attack showed itself.

The *quartan* returns every three days, that is to say on the fourth day, reckoning the day which the preceding paroxysm occurred.

Besides these, the more ordinary types of intermittent fever, there have been mentioned quintan, sextan, septan, octan and novan fevers; but during my long practice I have never seen them.

The principal types, however, the quotidian, tertian and quartan, present numerous varieties known as duplicated and reduplicated fevers.

The duplicated more common than the reduplicated, are those in which there are two paroxysms, occurring daily in the *quotidian*, every two days in the *double tertian*, and every three days in the *double quartan*.

As to the reduplicated fevers—there is in the *double tertian* one paroxysm every day, and in the *double quartan* two consecutive days with a paroxysm, and then one apyrexial day. Finally, in the *triple quartan*, the paroxysm occurs every day.

You no doubt, Gentlemen, expect me to explain the difference between quotidian fevers and those of the double tertian and double quartan types, as in all of them there is a daily paroxysm. The difference is this: in the quotidian, all the paroxysms are nearly identical with each other: in the double tertian, the paroxysm of the third day resembles that of the first day in form, intensity, duration, and hour of occurrence, and the paroxysm of the fourth resembles that of the second: in the triple quartan, the paroxysm of the fourth day resembles that of the first, the paroxysm of the fifth resembles that of the second, and the paroxysm of the sixth resembles that of the third day. For example, suppose a double tertian, the first day's paroxysm of which began about noon, and continued eight hours, while the paroxysm of the second day commenced only two hours later, and lasted only seven hours: the paroxysm of the third day, like that of the first, will begin also at noon, but will not terminate till 8 o'clock, while the paroxysm of the fourth day will begin at two o'clock, and will not be ended till nine o'clock, like that of the second day.

A remark, the full value of which you will understand when you have to treat a case of intermittent fever is—that the more the type is removed from the quotidian, the less amenable is the case to treatment. The quartan, therefore, is that which goes on for the longest time. The Latin imprecation, "*quartana te teneat*," for which it would not be difficult to find a French equivalent, shows, that the observation had in ancient times attracted the attention of the general community, as well as of physicians.

The same remark implies that the quotidian is the least obstinate: and that is the fact. Generally, a quotidian fever undergoes spontaneous cure without the intervention of the physician. This arises from quotidian fevers being seldom of marsh origin. They are met with everywhere, and at all seasons, at the commencement of certain pyrexiæ. In those countries in which intermittent fevers are endemic, most diseases in their earliest stage assume the quotidian type. I must add, however, that it is not unusual to see legitimate marsh fever take this type in young persons attacked for the first time: usually, however, in these cases, the fever soon becomes double tertian and then distinct tertian, thenceforth exhibiting all the characters of ordinary marsh fever.

The type seems to be derived much more from the nature of the miasm, or to speak more correctly, from the locality which it infects, than from conditions inherent in the individual.

Tours and Saumur, both situated on the left bank of the Loire, seem to me to present the same climatological and telluric conditions. Nevertheless, at Tours, tertian fevers are seldom seen; and the few cases of quartan fever which I there met with occurred in persons coming from Saumur, Rochefort, and other places where they had contracted the disease.

The following is an account of one of the most striking of the cases to which I am now referring. Fourteen soldiers living in barracks at Saumur went to Tours to give their evidence before a council of war. They had been hardly ten days in Tours, when nine of them were obliged to enter the hospital for *quartan* fever, the germ of which they had evidently contracted at Saumur; but all the fevers were tertian which I met with among the inhabitants of Tours and its environs.

An intermittent fever is called *regular*, when the paroxysms return at almost exactly the same intervals—*retarded* when the apyrexial period is prolonged—*anticipatory* when the apyrexial period is shortened.

In anomalous intermittents, the paroxysms may be *subintrant*—that is to say, one beginning before the other has terminated.

The diagnosis of an intermittent fever is certainly very easy; but no one, even the most experienced and talented, can pretend to that accuracy of judgment which would have been exacted by Galen who considered that the physician ought, from seeing the first paroxysm, to be able to say whether the fever was tertian or quartan:—"*tertianam quidem a quartana qui primo statim die, nescit distinguere, neque omnino medicus est.*" Although Galen has collected the differential signs which, in his opinion, ought to guide us, their value is too disputable to allow us to place any reliance upon them. Before we can know the type of the fever, we must wait till several paroxysms have occurred: nay, we must even wait to be sure that the malady really is intermittent fever.

Often, in fact, as I have just been saying, and as is observed particularly in countries where marsh fevers are endemic, some continued fevers commence as intermittents, though they soon assume their proper type. This is a point to which I directed your attention when lecturing on dothienenteria, in relation to the cases of two women we had in Saint-Bernard's ward, in whom typhoid fever had commenced with the symptoms of intermittent fever—in the one as a quotidian commencing abruptly, and in the other, first as a tertian, then as a double tertian, then as a remittent, and finally, as a well marked continued fever.

The subject is so essentially clinical, that I am not afraid of repeating the remarks I made on the occasion I refer to.

If your practice lie in a locality where marsh fevers are endemic, or among persons who have formerly inhabited such countries, I advise you to avoid concluding hastily that a fever is intermittent, unless the type be tertian or quartan: be likewise distrustful of appearances when the fever is double tertian, and be still more distrustful if it be quotidian. Before administering preparations of cinchona, which would fail in such cases, wait to see whether the type do not change. Should the fever prove to be continued, the intervals between the paroxysms will become shorter and shorter, and the paroxysmal manifestations will be less in this way : for example—if the shivering, chattering of the teeth, and general discomfort have continued an hour on the first three or four days, towards the fifth, sixth, or seventh day, it will not continue more than half an hour, and about the eighth or ninth day, the shivering will be quite transient. But while the paroxysms are less distinctively marked, the whole paroxysm will every day become more and more prolonged, the continued type will become more and more decided; and ere long, the disease will be exactly characterised. In addition to the manifestations of fever, there are symptoms which give a clue to the diagnosis. On interrogating, and attentively examining the patient, there are observable a certain number of phenomena which are not generally met with in marsh fever, and which belong to dothienenteria: I refer to the general softness of the pulse, vertigo, insomnia, and a feeling of discomfort, greatest in the interval between the paroxysms: then, there is the tendancy to diarrhœa, and the gurgling produced by making pressure over the right iliac fossa.

Intermittent fever symptoms may also supervene at the beginning of phlegmasiæ, pleurisy and pneumonia; and a still more prejudicial mistake may occur when the symptoms are those of inflammation of the pulmonary parenchyma, as they may lead us to believe that we have to do with one of the forms of pernicious intermittent fever.

On the other hand, in districts poisoned by marsh miasmata, you will see individuals suddenly seized with exceedingly violent symptoms of continued fever, which from their course and severity seem to indicate the onset of an attack of dothienenteria. After a certain time, these symptoms become complicated with shivering, which at regular intervals increase, and recur with characters more or less marked, the intermittent paroxysms following a gradation inverse to that which we have seen them presenting in the previously described cases, becoming quotidian, then double tertian, tertian, and even quartan.

You can now understand the full value of the hippocratic precept, to which I drew your attention a few minutes ago—not to interfere with intermittent fevers till there have been a certain number of well marked paroxysms.

By conforming to this rule, when you have to do with a dothienenteria clothed at its commencement with the symptoms of intermittent fever, you run no risk of employing inappropriate treatment, and of blaming the cinchona for having changed a generally mild into a severe malady. In an attack of mild synochus, such as we frequently meet with in Paris, assuming at first the aspect of an intermittent fever, and from which there is generally spontaneous recovery, we must not fall into the mistake of supposing that we have cured a marsh fever by small badly administered doses of cinchona, or sulphate of quinine, nor by some of the supposed febrifuges, such as kitchen salt or the bark of the *æsculus hipposcastanum*, remedies recently lauded, the apparent success of which is attributable to their having been employed in cases similar to those of which I have now spoken.

Finally, when we have to do with intermittent fevers, assuming at first the continued type, when we are waiting before we interfere, we must not suppose that we have reduced an incipient dothienenteria to a legitimate intermittent admitting of being cut short by cinchona.

Pernicious Intermittent Fevers.—What is the meaning of the term "Pernicious"?—Different kinds of Pernicious Fevers, such as the Algid, the Hot, and the Sweating; and those characterised by Coma, Delirium, or Convulsions.—They are usually of the tertian type.—They are Anticipating or Subintrant.—Colouring of organs, particularly of the Liver and Brain by pigmentary embolia.—The Pernicious symptoms may be due to Embolism.—Flagrant Insufficiency of the mechanical theory.—Masked Fevers.—Affections termed Neuralgic and Neurotic: Flux.

Gentlemen:—Regular intermittent fevers, like those of our two patients in Saint-Agnes's ward, are not generally of a serious character, except in so far as they lead to dangers inherent in that extreme cahexia which is induced by their prolonged duration.

It is otherwise in respect of *pernicious intermittent fevers*. They frequently involve immediate danger; and when not treated promptly and energetically, almost inevitably have a fatal issue.

They are of rare occurrence in Paris; though, as I have already said, they have been seen here rather more frequently of late years, in consequence of the disturbance of the soil necessitated by the improvements which have been going on in the town. They are very common in Algeria, and in some parts of Europe, such as in the environs of Rome, in the Pontine Marshes, as well as in some departments of France; they are still more frequent in the equatorial latitudes both of the old and new world.

Intermittent fevers are called *pernicious*, when there is a perturbation of the economy very greatly jeopardising the life of the patient within a few hours or days.

The pernicious symptoms of marsh intermittent fevers show themselves by exaggeration of the usual phenomena of the disease: when there is exaggeration of the shivering, the fever is called *algid;* when there is exaggeration of the febrile reaction, it is called *burning;* and when excessive sweating occurs, the term *sudoral* is employed. Functional disturbance of the organs essential to life is also met with; to pernicious intermittent fevers of this kind, the epithet *comitatæ* was applied by our medical predecessors.

I may now say in a word, to prevent the necessity of recurring to the topic, that the *pernicious* element of the fever depends less upon the disturbance excited in the economy by the affection of a particular organ, than upon the essential nature of the disease. It does not consist in the intensity of the functional disturbance of this or that organ, but in the insidious approach of imminent death—the malignity, the veritable, inherent, protopathic malignity with which in nearly every case the attack at once declares itself. The truth of this remark is shown by the fact, that the extent of this danger is far from being proportionate to the importance of the organ specially affected: this we see in pernicious intermittents characterised by cardialgic and dysenteric symptoms, and still more in the algid, burning, and sudoral forms just named, which do not seem to be concentrated in any particular organ.

Pernicious intermittent fever assumes the most diversified forms, which, I repeat, are characterised either by exaggeration of one of the usual phenomena of the disease, or by functional disturbance of some organ. The most common forms are the *algid* and the *sudoral*.

In algid fever, the cold continues from the beginning to the end of the paroxysm. It sets in with shivering of much more than ordinary violence, which rapidly increases in intensity: it lasts for a good many hours: the temperature of the body is really and obviously lowered: the tongue becomes icy: the skin when pinched retains the fold made in it, as it does in the algid stage of cholera-morbus. The thirst is intense; and the anxiety extreme. The face has a cadaveric expression:—*cadaveris imaginem refert,* says Borsieri. There is no acceleration of the pulse; and if the symptoms subside, the patient very slowly regains his heat.

In the sudoral fever—the *febris diaphoretica* of the ancients—sweating commences at a stage rather earlier than usual, and soon becomes so profuse as to bathe the surface of the body. Coincident with this cold sweat, there is a rapid, small, weak pulse, accompanied by quick painful breathing. The fingers seem as if macerated: the countenance is livid. The coldness is so great, that it is necessary to warm the patient, who may die in the first paroxysm. Should he emerge from it, his state of physical and mental prostration is extreme.

Among the forms of pernicious fevers called *comitatæ* by the older physicians, we have the comatose, soporose, apoplectic, lethargic, delirious, convulsive, tetanic or epileptic, syncopic, cardialgic, hemorrhagic, petechial and scorbutic, peripneumonic and pleuritic, gastralgic, hematemesic, choleraic and dysenteric. These terms are used to indicate the particular system of organs specially implicated, whether, for example, it be the nervous, respiratory, or digestive systems. They sufficiently point out the nature of the symptoms which characterise these different forms of fever.

In the *pernicious comatose fever,* the most striking phenomenon is a somnolence, which, showing itself at the beginning of the cold stage, or with the hot stage, goes on progressively augmenting during that second period up to the beginning of the sweating stage, being at last the profound stupor called carus. If an attempt be made to get the patients out of this state by giving stimulants, they open their eyes, but immediately shut them, moaning plaintively like persons roused out of their first sleep. In certain cases, the state to which I am referring is a veritable lethargy: in others, it is like an apoplectic stupor; and may be mistaken for apoplexy, if no notice be taken of the intensity of the fever, the heat of the skin, and the acceleration of the pulse, and if the duration of the initiatory shivering be not taken into account: the symptoms are scarcely so violent as in true apoplexy. The comatose symptoms gradually disappear within a time proportionate to the duration of the fever paroxysm, whether that be eight, ten, twelve, fifteen, or twenty-four hours. The patients then begin to take cognisance of the external world, and are amazed at what has occurred to them, of which they do not retain the slightest recollection. Though sometimes continuing to have a drowsy tendency, they seem completely restored to health, till the moment of the commencement of a new paroxysm, the recurrence of which will be more or less distant according as the fever is quartan, tertian, or quotidian.

The *delirious form* is characterised by a delirium, which, often announced by hallucinations, likewise shows itself at the beginning of the shivering, increases in intensity during the hot stage, and terminates with the termination of the sweating stage.

In *pernicious convulsive fever*, the convulsions are generally at once tonic, clonic, and epileptiform; but sometimes, though seldom, they are exclusively tonic and tetaniform.

The *syncopal*, so wonderfully described by Torti, is perhaps the worst of all the forms of pernicious intermittent fever.[1] It is the worst, in this sense, inasmuch as sometimes there is produced a state of apparent death, subjecting the patients to the risk of being left to die, when they might have been saved by timely medication.

A station-master of the Avignon railway, subject for some time to paroxysms of intermittent fever, had repeated fainting fits. The syncope on the last occasion was so complete, that the absence of pulse led to the belief that life was extinct. Upon what seemed evidence of death, the body was taken to the autopsy theatre. It had been there for only a few hours, when it was the will of Providence, that a servant should require to enter. This lad hearing groans perceived the error which had been committed, placed the unfortunate individual again in his bed, and summoned M. Chauffard, physician to the hospital. Cinchona was immediately administered in large doses. The untoward symptoms ceased; and the patient regained his health.

Another individual, having fallen into a faint, was likewise accounted as dead, and had the face covered with a sheet: M. Chauffard, when making his examination, observed, that although the radial, axillary, and carotid arteries had ceased to beat, some slight movements of the heart were still perceptible. He immediately ordered a quinine lavement: the patient was saved.

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[1] TORTI.—Therapeutica Specialis ad Febres Periodicas Perniciosas. Nova editio, curantibus Tombeur et Brixhe: 1821.

In some cases, the fainting is preceded by acute precordial pain, called *pernicious cardialgia.* It generally begins abruptly, without any apparent cause, the patient falling down in a state of exhaustion, induced either by a movement made in attempting to change his posture, or sometimes even by merely moving his arms. The pulse becomes small, accelerated, difficult to be felt, and then entirely ceases. The eyes are hollow; and, as in the cases I have laid before you, the person is apparently dead. Death may occur at any time subsequent to the first paroxysm.

Epistaxis, hæmaturia, a more or less abundant petechial eruption, covering a greater or less extent of surface, accompanied by a small frequent pulse, characterise *pernicious hemorrhagic fevers,* which are petechial and scorbutic.

The symptoms commence with shivering, followed by a more or less severe hot stage, which is always accompanied by precordial anxiety, and sometimes by pain extending from the loins to the back, and to the pit of the stomach.

Peripneumonic pernicious fever, or, to borrow from Torti and Morton the more appropriate name, *pernicious catarrhal fever,* is characterised by pulmonary symptoms. The respiration is difficult, embarrassed, and disturbed: the face is turgid, and the eyes are injected: the forehead and chest are covered with sweat. In addition to these external signs of suffocative catarrh, there exists copious expectoration of a mucous bloody character, like that seen in certain cases of pulmonary apoplexy; and upon auscultation, crepitant and fine subcrepitant râles are heard in all parts of the chest.

Pleuritic pernicious fever declares itself by a stitch in the side, in some cases acute and poignant, and in others obtuse: the pain is increased by the respiratory movements, which are short and embarrassed. The pulse is small, hard, and often unequal. On percussion and auscultation, it is found, that in the corresponding side, there is pleuritic effusion, which is generally absorbed during the interval between the paroxysms.

In *cardialgic pernicious fever,* the pain at the pit of the stomach is so dreadful—so ferocious, to adopt the epithet applied to it by Borsieri[1]—that the patients utter terrible cries. Vomiting, at first mucous, then bilious, supervenes. There are other cases in which there is hematemesis; and in such cases, the disease is said to be a pernicious *hematemesic* fever.

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[1] Borsieri:—Institutiones Medicinæ Practicæ. Lipsiæ: 1825—1826.

When the intestine is attacked, there may be an alvine flux resembling the water in which raw meat has been washed, and as profuse as the discharge from the bowels in cholera. This form of the disease is called *pernicious choleriform fever*; or *pernicious dysenteric fever* when the flux is a sanguineous secretion. In both of these forms, there is great prostration of strength.

Gentlemen, it is essential to bear in mind, that all these different forms of pernicious fever, however various, have characteristics in common.

In respect of type, they are generally tertian, sometimes quartan, exceptionally quotidian. Cases are likewise mentioned in which the type was remittent; and when the rigors are recognised with difficulty, continued fever may be simulated.

Though generally tertian or quartan, pernicious intermittent fevers do not assume these types till after several paroxysms have occurred. However, in countries where the poisonous miasmata have great power, as in Africa, or in individuals who after living in these countries, come to other countries, carrying with them the morbid germ, the fever may at once become pernicious, and carry off the patient in its first paroxysm. At the autopsy, in these cases, as in the others, the spleen is found to be enlarged, softened, pulpy, and sometimes ruptured.

Generally, I repeat, the disease does not so rapidly assume its pernicious character. The patient has first had many regular paroxysms of fever, which after a certain time change their character. The different stages are prolonged beyond their ordinary duration and leave the patient in a state of debility, contrasting remarkably with the amelioration—the return to almost perfect health—which followed the previous paroxysms. Finally, there supervene the morbid phenomena which mark the pernicious character of the malady.

When the symptoms from the first paroxysm in which they show themselves, do not indicate that the case will have a fatal termination, they follow the course which I have pointed out to you, beginning, that is to say, with the cold stage, increasing with the hot stage, and gradually decreasing from the commencement of the sweating stage, till they at last spontaneously cease. It is very necessary to be aware that this is the course of events; for if, mistaking the nature of the phenomena, the physician is eager to combat the symptoms—the coma, convulsions, or delirium, for example—by local or general bleeding, or by any other measure which is at least useless if it be not dangerous, he will, from the moment that calm begins to show itself, take credit for having obtained a fortunate result, which his illjudged intervention has only not prevented. At the very time that he is congratulating himself upon having achieved a success, the patient may be carried off by a paroxysm which might have been averted by appropriate treatment.

We must also beware of not being misled as to the nature of the malady, when the paroxysms of pernicious fever are separated by intervals shorter than those of regular intermittents.

When the paroxysms are prolonged beyond their usual duration, the fever is called *anticipatory* or *subintrant*:—*anticipatory*, when, for example, a first paroxsym having commenced at noon, the succeeding paroxysm begins six hours earlier; and *subintrant*, when the second paroxysm begins before the first has terminated, the paroxysm imbricating in such a manner as to prevent the patient recovering from the shock of one attack before he has to sustain a second. Were it for this reason alone, anticipatory, and still more subintrant intermittent fevers, irrespective of any complication, demand the active intervention of the physician. But although we may, or rather ought, to adopt the precept of the older physicians,—to wait, in a regular intermittent, till several paroxysms have occurred—we must beware of delay when the stages exceed their normal duration. In such cases, even when the stages have not presented anything unusual, act without loss of time, particularly if you perceive any disturbance of important organs, for then danger is imminent, and there is no time to lose.

I must not omit to mention a pathogenic theory promulgated in Germany by men whose works I exceedingly esteem. The theory to which I refer attributes the pernicious symptoms to *pigmentary embolia*. For the sake of those among you who are not acquainted with these views, I think it will be useful to enter somewhat into detail.

In the bodies of persons who have been under the influence of marsh miasmata and have succumbed during a paroxysm of pernicious fever (intermittent, remittent or continued), we frequently discover certain lesions of the liver, that organ presenting a steel-grey or blackish colour—sometimes a chocolate colour. This change of colour is due to the accumulation of pigmentary matter in the vascular apparatus of the organ: the liver is pigmented.

The spleen presents exactly similar lesions: it has a sombre or bluish-black hue, which is either uniform or disseminated in patches. In its interior, we find a still greater quantity of pigmentary matter than in the liver.

We also always find black pigment in the capillaries of the lungs.

It is likewise found in the brain, where it is still more easily detected, from its impinging upon the white colour of the organ. The cortical substance then assumes the colour of chocolate or graphite, while the medullary substance is not in any degree modified; or at least only in those cases in which there is a great amount of pigment. Under the microscope, the capillaries are to be seen full of granules and black particles, sometimes uniformly distributed, and sometimes accumulated in masses.

What then is this pigment? What is its mode of formation within the organism? It is a matter of a deep black colour; or, it is ochrish brown; or, in exceptional cases, it is yellowish red. It is formed to a great extent of granules, with which are found a small number of true pigmentary cells containing within them a greater or less number of black granules. The pigmentary granules are amorphous; but the agglomerations which they form are sometimes irregular, and sometimes cylindrical, from their being moulded in the interior of the vessels.

The pigment is the result of the transformation of the hematine; and this transformation may take place *anywhere, even external to the vascular system.* I beg that you will remember this fact. The metamorphosis is purely physical, or is, in other words, absolutely independent of the vital laws.

So much for fact: let us now attend to the theory.

The pigment is manufactured in the spleen, which organ decomposes the blood-globules, and transforms their hematine into pigment. The pigment thus formed passes into the vena porta, then into the liver, then into the vena cava, and then into the heart, whence it is carried into the general circulation. Here, we find ourselves brought back to the old atrabiliary doctrine; but also to a doctrine which is highly scientific and thoroughly armed.

Intermittent fever determines hyperemia of the spleen: under the influence of this hyperemia, the spleen manufactures a very large quantity of pigment. "In fact," says Frerichs, "in the normal state, the blood passing at once from the narrow capillaries into the venous cavities, flows slowly, and sometimes stagnates at certain points: then forming agglomerations of blood-corpuscles, it is, little by little, metamorphosed into pigment. In the hyperemia of the spleen consequent upon intermittent fever, the stagnation is exceedingly marked, and *the result is the formation of masses of pigment.*"[1]

Frerichs is likewise of opinion, "that the pigment is developed at the expense of the blood which remains in the venous sinuses: the cellules, spindle-like and clubbed, of this pigment are formed from the internal parietal epithelium of the sinuses impregnated with decomposed hematine: the globular cellules are white blood-corpuscles charged with molecules of colouring matter: the pigmentary masses are detached fragments of concretion."[2] I have quoted the exact words of Frerichs, so that I might be certain to place correctly before you the German theory.

This extensive destruction of blood-globules by the spleen directly, necessarily impoverishes the blood; and this explains the anæmia which is so common a sequel of marsh fevers.

The excessive production of pigment will lead to a series of consequences, both of an organic and functional character, depending upon two causes entirely of a mechanical nature—the crowded state [*encombrement*] of part of the capillary system, and the damming [*barrage*] of the circulation. Indeed, Frerichs admits that the masses of pigment are stopped in the capillaries of organs, and there constitute a species of embolism.

To this mechanical doctrine, which is essentially only an application of the doctrine of embolism to the theory of pernicious symptoms, a doctrine far too superficial, and even in contradiction to the facts observed by the authors who support it, Dr. Peter and I make the following objections:—

This crowding [*encombrement*] induced by embolism must be, and in fact is, first produced in the capillaries of the liver, the organ first in the course of the blood returning to the spleen. The obliteration of a part of the hepatic capillaries, and the consequent stasis of the blood, produce disturbance in the process of hematosis, because the liver is a hematopoietic organ; and Frerichs has very nearly adopted the belief that the gastro-intestinal hemorrhages, the attacks of profuse diarrhœa, and the vomiting, are the ultimate consequences of functional disturbance of the liver.

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[1] FRERICHS: Traité des Maladies du Foie; traduit de l'Allemand par Louis Duménil et J. Pellagot, p. 497. Paris; 1866.

[2] FRERICHS: Op. cit.

However, some of the pigment traverses the liver, passes into the right side of the heart, and reaches the lungs, where it ought to induce pigmentary crowding [*encombrements pigmentaries*]; but it would appear that this mischief-working material causes no bad effects, for Frerichs observed that the appearances found at the autopsies do not allow us to attribute the dyspnœa, or other functional disorders of the respiratory function, to pigmentary obstruction of the capillaries of the lungs. As you know, the capillaries of the lungs are smaller than those of any other organ. One cannot, therefore, understand how it is that the pigment which has been able to pass through the hepatic capillaries, which have a larger diameter, should not be stopped in the pulmonic capillaries which have a smaller diameter: but this is not the only absence of natural consequence involved in the iatro-mechanical system.

It appears then, according to this theory, that, notwithstanding seeming improbability, pigment traverses the lungs, reaches the right side of the heart, whence it is discharged over the whole organism: hence results, *melanæmia*, that condition of which you have heard me speak, which explains the peculiar colour of the face in marsh cachexia. We ought to find pigmentary embolism in every organ; but this is not the case, for the brain and kidneys almost exclusively enjoy this unfortunate privilege.

Frerichs says:—"In the most minute capillaries of the brain, principally in those of the cortical substance, there collect particles of pigment which have traversed the vessels of the liver and lungs, while none have been arrested in their passage through these last-named organs. These mechanical impediments to the circulation, often occasion numerous lacerations of the vessels and capillary apoplexies."

Albuminuria is the indication of functional disturbance of the kidneys which is met with.

A host of objections present themselves to the embolism or blockade theory of pernicious intermittent fevers. Every embolism necessarily supposes an aggregate too voluminous to traverse the capillaries of organs, particularly those of the brain. But as anatomy shows that the capillaries of the brain and lungs are the smallest in the body, it follows, that the pigmentary aggregate must in the first instance encounter a material impossibility to traverse the capillaries of the lungs. There is no escape from this dilemma: the pigment either has been arrested in the pulmonary capillaries, there producing embolism, all the pigment which has traversed the liver being stopped in the capillaries of the lungs; or the pigment has found its way through the pulmonary capillaries, in which case, it must have passed through the capillaries of all the organs, because they are either as large or larger than those of the lungs.

It is, however, impossible to deny the existence of pigmentary colour in the organs, and the presence of masses of pigment in their capillaries. A fact does not admit of being denied; but then this fact can be explained in a perfectly natural manner, that is to say, by the pigment being formed *in situ,* and being a consequence, not a cause. Thus, Frerichs himself admits that the pigment may be produced in any situation, even external to the capillaries. Pigmentation, then, is a physical phenomenon, arising simply from the destruction *in situ* of a certain number of globules by the stasis of the blood in the vessels. This stasis is itself a result of repeated visceral congestions, which are passive, and proportionate in intensity to the intensity of the fever. The blackish colour of the organs, particularly of the nervous centres had been described by the authors who had studied pernicious fevers, particularly by Maillot and Bailly; but they attributed this colour to congestion connected with the condition existing during the paroxysm. Is it not more simple to consider the "pernicious" symptoms as the effects of a sanguineous *raptus* in the nervous centres and their envelopes;"[1] and to see in the pigmentation the physical result of a consecutive alteration of the blood *entirely cadaveric,* and which has taken place *in situ* from the local destruction of globules crammed into the capillaries distended during life? This doctrine so simple, and so naturally present to the mind, is certainly more true, or at least more like truth, than that which invokes the migration of embolia, which, as I have shown, is physically impossible.

There is also a purely clinical objection to this, viz. that the *intermittence* does not correspond with the *permanence of the lesion.* If the pigmentation be due to embolism, it ought to be persistent, for otherwise, there could be no embolism. Why should not the symptoms be as permanent as the lesion?

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[1] MAILLOT:—Traité des Fièvres ou Irritations Cérébro-Spinal Intermittentes. Paris; 1836.

Finally, there is the therapeutical objection involved in the all-powerful action of preparations of cinchona which is quite inexplainable by the pigmentary theory. How, in fact, are we to understand the sulphate of quinine dispelling, as it does, the pernicious symptoms, if their proximate cause be the blocking up of the vessels of the brain to remedy which condition cinchona has absolutely no power?

In my opinion, the pigment found in the vessels is formed in the place where it is found, and is the result of great and repeated congestions.

To sum up what I have said:—I look upon pernicious intermittent fever as a general disease producing many visceral congestions, particularly in the nervous centres.

From an organic point of view, there results from these congestions sometimes an alteration *in situ* of the blood-globules (pigmentation), or, sometimes, an interstitial hemorrhage. Hence result, looking at the case from a dynamic point of view, more or less severe functional disturbance.

Let me add, in conclusion, that there may exist pigmentation of the brain without pernicious symptoms; and also, pernicious symptoms without pigmentation. Frerichs admits this, with a loyal truthfulness to science which cannot be too highly praised. He says:—"If we compare the facts ascertained by microscopical examination with the symptoms observed during life, we will find, on the one hand, cases in which, notwithstanding the dingy colour of the brain, there has been no cerebral disturbance, and on the other hand, cases in which there has existed disorder of the brain, although the organ was free from pigmentation. In eighty cases of cephalic intermittent fever, this occurred six times. The older observers, including Lancisi, Sénac and Bailly,[1] made the same observation, which has been confirmed by Maillot[2] and Haspel.[3] It cannot, therefore, be doubted, that the cerebral symptoms already described cannot occur in intermittent fever unless melanæmia exist, and no other cause can induce these pernicious accidents.[4]

Gentlemen, under all the forms assumed by pernicious fevers, we find, either separately or united, three conditions, viz. pain, sanguineous congestion, and flux, characterising *masked* fevers, of which I have now to speak to you.

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[1] Bailly:—Traité Anatomico-pathologique des Fièvres Intermittentes Simples et Pernicieuses. Paris; 1825.

[2] Maillot:—Op. cit.

[3] Haspel:—Maladies de l'Algérie. Paris; 1850.

[4] Frerichs:—Op. cit., p. 503.

Pernicious fevers—those at least which the authors of past ages called *comitatæ*—are, in fact, masked fevers [*fièvres larvées*]. You must not use the epithets *masked* and *latent* as synonymous.

A latent is in reality a hidden disease: if it be a pleurisy, for example, it does not reveal its existence by any external symptom. The patient has none of the ordinary symptoms of inflammation of the pleura: he has neither stitch in the side, cough, nor dyspnœa: and when the physician discovers the existence of the affection, it is only from the physical signs furnished by auscultation and percussion.

A masked disease [*maladie larvée*] far from concealing, very distinctly manifests, its existence; but in manifesting itself, it puts on the mask [*induit larvam*] of another disease having little or no real similarity with it.

Thus, as I have already said, pernicious fever, in its delirious form, sometimes simulates brain fever, sometimes apoplexy or epilepsy, sometimes pneumonia or pleurisy, and sometimes cholera or dysentery. However, in borrowing from these diseases some of their principal phenomena, it retains other characteristics by which it can be recognised: the former, moreover, want many essential characters which belong exclusively to the affections, the names of which, for lack of better, are employed to designate the different kinds of pernicious fevers.

Let me explain myself:—The pernicious epileptiform intermittent borrows from epilepsy its convulsions; but then, the convulsions differ from those of epilepsy. In the pernicious pneumonic intermittent, the crepitant râles and sanguinolent expectoration remind one of the symptoms observed in certain forms of pneumonic catarrh; but without taking into account the appearance of the sputa, there are notable differences between the thoracic symptoms of the two affections. Pernicious dysenteric intermittent has bloody stools as a character in common with dysentery; but the tenesmus and glairy excretions, of so much diagnostic value in the latter, are wanting in the former. Observation of the progress and features of the disease enables one unhesitatingly to recognise its nature.

Though most pernicious fevers are masked fevers, it does not necessarily follow that masked fevers are pernicious: thus, the paludal diathesis very commonly masks itself as neuralgia, and under the form of certain neuroses. A woman, who lies to-day in bed 17 of our Saint-Bernard's ward, presents one of the most common types. This patient, aged twenty-six, tells us, that about four years ago, six months after the birth of her first child, whom she suckled, she was attacked with facial neuralgia: it particularly affected the eye, which, with each paroxysm of pain, became the seat of very acute congestion, and the source of a profuse flow of tears. The paroxysms recurred regularly every three days: they were announced by rigors, during which the pain supervened; it went on increasing during the hot stage, diminished during the sweating, and completely ceased after some hours. This attack of intermittent neuralgia resisted the most energetic treatment for many months. The patient was ultimately cured.

Three months afterwards, similar symptoms recurred under similar circumstances, that is, after chidbirth and during lactation; but with this difference, that the neuralgia in place of being suborbital, as in the first attack, was occipital.

Though to-day we only find pain in this case, the three elements characteristic of masked intermittent, pain, fluxion, and flux, were very manifest in the first attack.

The pain was seated in one of the branches of the fifth pair of nerves: the fluxion, seated in the mucous membrane of the eye, attained a degree of congestion sufficiently violent to simulate formidable ophthalmia: the flux was a profuse secretion of tears.

The nerves of the fifth pair are those usually affected in masked fevers. The neuralgia returns at regular intervals, assuming the different types of intermittent fever, the quotidian, tertian, double tertian, quartan, double quartan, and triple quartan. Whatever be the seat of the neuralgia, whether the neuralgia be sciatica, gastralgia, or enteralgia, it follows this rule.

There are also, as I have just stated, certain neuroses which constitute masked intermittents. Among them, is *spasmodic cough*, which in some subjects exposed to marsh misasmata recurs in paroxysms daily at the same hour, without being accompanied by expectoration or any morbid pulmonary symptom, and which yields in a remarkable manner to the treatment generally efficacious in cases of a similar nature.

There is also a species of *asthma*, which likewise recurs at regular intervals, and yields to the same therapeutic indications.

Let me also mention, that these remarks are applicable to certain cases of *megrim* and *hiccough.*

There are cases in which *insomnia* recurs every two or three nights, unaccompanied by fever, and not preceded by rigors, which are evidently of the same nature, and are cured by similar treatment.

The same may be said of *periodical fluxes,* more or less profuse, from the nasal fossæ, uterus, and intestines, sometimes simply mucous, but at other times sanguinolent, unattended by any other morbid symptom, and yielding to medication similar to that which is useful in marsh fevers.

You are now in a position, Gentlemen, to understand more easily the mechanism of masked pernicious fevers. You have seen the presence of pain, fluxion, and flux in neuralgia. Transfer these phenomena to another nervous apparatus, to the ganglionic nerves, or to the mixed nerves, as, for example, to the pneumogastric; and, bearing in mind the beautiful experiments of Claude Bernard,[1] you will at once understand the great functional disturbance which may be induced in these nerves, by morbid changes taking place in them. Suppose that there existed in the lungs or intestines, phenomena similar to those you have just seen in the eye, you would at once understand the dyspnœa, crepitant râles, and expectoration, or the pain in the intestines, the excessive flux from the gastro-intestinal mucous membrane and glands which discharge their secretions upon it. Transfer to the brain or spinal marrow the element of pain and fluxion which we have just been considering in the suborbital nerve, and see whether you will not have nervous phenomena depending upon the pain itself, or upon the fluxion, such as delirium, convulsions, stupor, and coma.

We may in this way pretty easily explain the very varied symptoms of both simple or pernicious masked fevers: in this way, confirmation is given to an opinion already held by others, and in which I completely concur; viz. that intermittent fevers, under whatever forms they may present themselves, ought to be classed with the neuroses.

There are numerous reasons, Gentlemen, for adopting this view. The suddenness of the invasion of the disease, and its rapid disappearance—the violence and likewise the fleeting character of the symptoms which characterise it—the terrible disorder which supervenes throughout the whole economy, a disorder which, in pernicious fevers, cannot recur without greatly endangering the life of the patient. Then, there is the deceitful feeling of security during the interval between even the most violent paroxysms, the wonderful facility in mastering a disease manifesting itself in such frightful forms. Do not all these circumstances, Gentlemen, lead us to adopt the idea that it is a neurosis? Then, again, when we see the most common pernicious masked fever generally accompanied by symptoms which even the most incredulous are forced to regard as nervous. When in these same pernicious fevers, it is so easy to take account of the symptoms from the disorder which they occasion in the nervous centres or ganglionic uerves, we are compelled to regard all forms of intermittent fever as so many different forms of neurotic disease.

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[1] BERNARD, Claude:—Leçons sur la Physiologie et la Physiologie du Système Nerveux. Paris; 1858.

When a neuralgia is intermittent, we must not conclude that it is necessarily a masked fever. In summer, for example, we not unfrequently meet with cases of violent neuralgia in which the attack begins every morning and ceases every evening, recurring in this manner for six, seven, or eight days in succession. This kind of neuralgia—*solar neuralgia*—is met with in the most salubrious countries, wholly irrespective of any influence of paludal miasmata.

When intermittence is the predominant character of a masked fever, it is important, with a view to the diagnosis, to inquire into the antecedents of the patient. Intermittence is very common in localities where marsh fevers are endemic: elsewhere, it is of rare occurrence; and in Paris, it is very seldom met with, except in individuals who have lived for some time in marshy countries, whether they have or have not had regular intermittent fevers.

The fact that the patient has sojourned at some former period, more or less distant from the appearance of the symptoms, in a marshy country, renders it probable that the disease is intermittent fever; and this presumptive diagnosis becomes almost a certainty, if there exist engorgement of the spleen, along with that peculiar tint of skin which indicates a state of cahexia. The nature of the disease does not admit of any doubt when it yields very readily to the treatment which is successful in paludal affections.

Treatment by Cinchona (according to the Roman, English, and French systems) and by Arsenic, and the Method of Dr. Boudin.

Gentlemen:—I have still to speak to you on the subject of treatment. I need not tell you that the basis of the treatment consists in administering cinchona, and its derivatives, quinine and the sulphate of quinine. Every one knows, that by means of these precious remedies, intermittent fevers are cut short. But every one does not know, that to cut and to cure an intermittent fever are not synonymous terms: this is a fact with which many physicians even do not seem to be acquainted.

It is necessary to administer cinchona according to a plan, so as to obtain the expected results. In my opinion, the best system to follow is that which has been lauded by Bretonneau, and is called the *French method*—a method which I have endeavoured to improve; and which is a happy combination of the methods of Torti and Sydenham.

Torti's method, likewise called the *Roman method* (from its having been first adopted by the Jesuits of Rome, who derived it from their brethren of Lima), consists in giving the cinchona, immediately before the paroxysm, in one large dose.

This method has inconveniences which were fully pointed out by Sydenham. When administered immediately before the paroxysm, it is often vomited: this inconvenience was recognised by Torti himself, who, to avoid it, sometimes gave the medicine after the paroxysm. On the other hand, as has been shown by the experiments of Bretonneau, the impending paroxysm which it is intended to stop, is often rendered more violent and painful by the cinchona.

To avoid these inconveniences, Sydenham and Morton directed the cinchona to be given at the longest possible interval before the anticipated paroxysm, commencing the remedy, consequently, as soon as possible after the termination of the preceding paroxysm. The following is Sydenham's formula:—

Take of Powder of cinchona, 32 grammes;
Syrup of roses and pinks, q. s.

This electuary is divided into twelve doses, one of which the patient is directed to take every four hours, commencing at the close of the paroxysm.

He was also in the habit of prescribing a wine of cinchona containing thirty-two grammes of the powder in two pounds of common red wine.

He ordered the patient to take, in a manner similar to that now described, from eight to nine tablespoonfuls of this preparation.

You have seen me on several occasions, and recently in a case of obstinate quartan fever, have recourse to an electuary very similar to that of Sydenham, the only difference being, that, in place of the syrup of roses and pinks, I used the conserve of roses, or the syrup of bitter oranges, which latter I prefer, as it has the advantage of masking the disagreeable taste of the cinchona.

It is undoubtedly an important rule to give the cinchona at a period as long as possible before the coming paroxysm. The reason is very simple. The active principle of the remedy being neither volatile nor diffusible, is absorbed slowly, and requires a certain time to produce an effect upon the economy. When the quantity administered is not more than an ordinary dose, this time is at least eighteen or twenty-four hours: large doses take effect in from six to twelve hours.

Hence it follows, that the best method of treatment consists in employing a large dose of cinchona divided into three or four portions, giving these portions at very short intervals, and commencing the administration as long as possible before the coming paroxysms is due.

This, which is Bretonneau's method, is a happy combination, as I have already remarked to you, of the methods of Sydenham and Torti; and is that which you have seen me employ. By following it, you will obtain results much more complete and sure, from far smaller quantities of cinchona than would otherwise be required. Fifteen grammes of cinchona administered at once are generally sufficient to suppress a paroxysm of regular intermittent fever; but thirty grammes, that is to say, twice the quantity, given within five or six days in the apyrexial intervals, will fail to produce that effect. We must not, however, adopt to the letter the precept of Torti and Bretonneau. By a single dose [*une seule dose*] we must understand that the entire quantity of cinchona prescribed is to be taken within a very short space of time, within one, two, or three hours at most; for some patients cannot easily tolerate at once fifteen grammes of cinchona, or even eight grammes, the dose of the powder recommended by Torti,[1] and that which I usually prescribe. This remark is equally applicable to the sulphate of quinine.

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[1] Torti:—Therapeutice Specialis ad Febres Periodicas Perniciosas.

In simple legitimate intermittents, of which alone I am now speaking, I prefer powder of cinchona to the sulphate of quinine.

It has not been proved to my satisfaction that quinine, (still less its salts such as the sulphate), possess all the virtues of the bark of cinchona: on the contrary, experience has taught me, that although the bark acts more slowly, it acts more surely and more thoroughly, if I may so express the fact, that the salutary effects are more lasting. Does this arise from the bark containing in addition to that which we regard as its active principle, other principles with which we are not acquainted? Does this depend upon the crude drug yielding slowly its quinine, so as to admit of its being assimilated, while this is not the case with the sulphate of quinine, a part, the greater part of which perhaps, is eliminated with the urine? These are questions which I cannot answer. Nevertheless, experience has proved the correctness of the statement I have now made.

Moreover, for economical reasons, which may perhaps appear trivial to some savans, one superiority of the bark will be appreciated by physicians, particularly by those whose practice lies in the country among the comparatively poor, to whom greater cheapness will render the bark very preferable to the sulphate of quinine.

When it is necessary to give daily as much as a gramme of the sulphate of quinine to suppress a paroxysm of intermittent fever, similar results may be obtained, and that too with more certainty, from eight grammes of cinchona, that is to say, from a dose only one fifth the price of the equivalent dose of sulphate of quinine. In a word, then, when I wish to cut short a regular intermittent fever, I give, immediately after the termination of the paroxysm, eight grammes [124 grains] of the powder of yellow cinchona, either at once, or in two portions at the interval of half an hour, in a cup of tea or coffee.

This dose rarely fails to prevent the recurrence of the impending paroxysm. But although the disease is arrested, it is not cured, the arrest being only temporary, and the patient retaining some slight reminders of his malady, such as an increase of temperature with feelings of general discomfort, or—and this is more usual—profuse sweating recurring on the days upon which the paroxysm was due. If the medication be abruptly discontinued, if the patient do not return to the use of the quinine, the symptoms soon reappear, first feebly and in a less decided form, but ere long with all their precise and positive characteristics. It is, therefore, important, even though there be no immediate return of the symptoms, to continue to give the febrifuge in the same doses, at the determinate intervals, for a period prolonged more or less according to the duration of the malady, according to its type, and particularly according to the patient being or not being resident where he has contracted the fever.

Torti, in simple intermittent fevers, gave eight grammes of the powder of cinchona immediately before the paroxysm was expected to begin: and he continued the administration of the medicine till the patient found himself quite well: day by day, however, he diminished the dose. This method of administration, adopted by Torti from the Roman Jesuits, and for that reason designated the Roman method or the method of the Fathers, is often excellent in the case of patients who have ceased to reside in the localities where intermittent fever is endemic, and who have not been long subject to recurrence of the paroxysms. It saves the patient from attacks of fever for ten or fifteen days, or even, some times, for a longer period; but it has not the power to prevent relapses.

Sydenham's method, called the English method, has a much greater power. Sydenham, as I have already stated, begun by giving immediately after the termination of the paroxysm thirty-two grammes [496 grains] of the powder of cinchona, which he gave in doses distributed at nearly equal intervals between the past and coming paroxysms. Eight or fourteen days later, according to the type of the fever, he repeated the same dose: he likewise recurred several times to the same medication, particularly if the patient had long had the fever, and had suffered in constitution from paludal influence. This method is much more efficacious than that of Torti: it is much more protective from relapses: but it has its inconveniences which I must now state.

Many patients cannot take so large a quantity of powder of cinchona without having vomiting, and still less without diarrhœa being induced. Then again, patients who have been long subject to the fever, have a recurrence of the paroxysm, after the lapse of some days, and before the sort of sacramental epoch fixed by Sydenham for the readministration of cinchona. One may, it is true, ward off the first of these inconveniences by giving a small quantity of opium along with the febrifuge powder; but the objection to the method remains undiminished when the patient is deeply imbued with paludal cachexia.

Gentlemen, recall to your recollection the woman who came from Guadaloupe with tertian fever of more than six months' duration. Sydenham's doses, to which were always added a little laudanum, cut short the malady and gave the patient a respite from attacks of fever for five or six days; but between the sixth to the seventh day, a new paroxysm announced to us, that it was the paroxysm and not the patient that had been cured. It then became necessary to resort to the *method of Bretonneau*, the *French method*, which I have modified to a certain extent, and in a manner which my illustrious master has himself adopted.

Bretonneau found that Sydenham's daily quantities were too large: that when there was involved in the question of dose the cost of a remedy so expensive as cinchona, or, and still more, the sulphate of quinine—and also, when it was remembered that intermittent fever was specially a scourge of the poorer classes,—the money value of the dose was a matter of great importance. He had learned from experience that Torti's dose of eight grammes was sufficient, provided it were administered at the longest possible interval prior to the coming attack, either all at once, or divided into two portions taken with an interval between the times of not more than some hours. Experience had likewise taught him, that by daily administering decreasing doses, according to Torti's plan, relapse was almost inevitable. In respect of dose, therefore, he adopted the Roman method: in respect of mode of administration, he adopted the method of Sydenham, that is to say, he administered, the febrifuge immediately after the paroxysm, renewing the administration, *always in similar doses*, at longer or shorter intervals, and extending over a considerable period of time, according to the rule of Sydenham.

Bretonneau's method, or the *French method*, as it is called, consists in giving eight grammes of the powder of yellow cinchona bark or one gramme [15½ grains] of the sulphate of quinine, in a single dose, or in two doses with a very short interval between them, as long as possible before the coming, that is to say, immediately after the past paroxysm. Five days of rest are allowed to pass, when the same dose of the remedy is again prescribed; and then, it is repeated once in eight days for a month.

When the fever has been of very long standing, it is necessary to continue the medicine for a very long time, augmenting the dose if necessary, as the treatment proceeds. From the second month of the treatment, the cinchona is taken at successive intervals of ten, fifteen, twenty-five, or thirty days; and according to such a plan as will secure the patient against relapses, a result obtained with much less certainty when Sydenham's method is rigorously followed.

During three years which I passed at the hospital of Tours, following Bretonneau's clinic, I only saw once this method fail to cure intermittent fever. However, since I have been at the head of a service in a Parisian hospital, I have repeatedly failed to cut short perfectly regular intermittent fevers, though I employed exactly the formulæ of my illustrious master. The first paroxysm following the administration of the cinchona was postponed, much diminished in severity, or sometimes even suppressed, but the second, or at least the third paroxysm, reappeared in a more or less modified form: here then was a sufficiently serious inconvenience. I avoided it by adopting the method which I have pointed out to you.

Immediately after the termination of the paroxysm, I cause the patient to take eight grammes of the yellow bark, or one gramme of good sulphate of quinine, either in one dose, or in two doses with an interval of one or two hours between them. For a day, I leave the patient without medicine; and on the third day, I give him the third dose of the medicine, administering it either all at once, or at twice, the taking of the second rapidly following the taking of the first portion of the dose. I then allow an interval of three days, four, five, six, seven, or eight days, and finally, for one or two months, I recur every eight days to the same medication, *never diminishing the dose*. Let me add—and the fact is important—that the medicine ought always to be given at a meal.

Bretonneau's method and my own are in reality only modifications of the method of Sydenham.

Gentlemen, you have sometimes witnessed in our wards, the failure of a treatment of which I have just now been proclaiming the infallibility.

You can recall to your recollection the young men who returned to France, reentering civil life after having made the Crimean, African, and Italian campaigns, who when admitted to our wards, suffered from dreadful cachexia, enormous engorgement of the spleen, and infiltration of the extremities. They refused to take preparations of cinchona, because, as they said, cinchona had done them harm, and had not cured them. I have frequently called your attention to these patients, requesting you to mark the happy influence of the medication which I was about to institute. These are the circumstances under which Sydenhamian doses ought to be unhesitatingly recommended: these are the cases in which the powder of cinchona resumes its old claim to superiority, and leaves far behind it the sulphate of quinine: these are the cases in which my method, scrupulously followed, and aided by the use of ferruginous medicines, produces results so evident as to astonish and convert even the most incredulous.

Very recently, you have seen me treat in a similar manner a man who occupied bed 9 in Saint-Agnes's ward. In that case, I began by giving an emetic and the purgative decoction of cinchona (of which I shall have forthwith to speak to you): next day, between the two paroxysms, I made him take thirty grammes of Sydenham's electuary (the formula for which I have given you), taking care to administer a drop of laudanum with each dose, so as to prevent vomiting and particularly diarrhœa. I recurred to the same dose, in the first instance after one day, then after three, four, five, six, seven, or eight days of interval, prescribing always thirty grammes of cinchona, with also, at each meal, a table-spoonful of the syrup of the ammonio-citrate of iron. You have seen that during the six weeks of the patient's residence in hospital, he has not had the slightest feeling of a return of the fever, that the spleen has rapidly diminished in size, that his colour has been natural, and the digestive functions have been reestablished in their normal state: you have also seen that this man so exceedingly dispirited, so distrustful of the curative powers of the preparations of cinchona, obtained a rapid cure, which was due as much, or perhaps more, to the method of employing the remedy as to the remedy itself.

If the most intelligent administration of cinchona be abruptly abandoned after some days, the fever returns: under such circumstances, it is necessary to begin the treatment exactly as it was undertaken in the first instance.

When, in accordance with other methods of administration, a small dose of cinchona or sulphate of quinine is given daily, the fever is modified and is sometimes cured; but the cure is accomplished with more difficulty, and less certainty: acute gastric pains soon supervene, under whatever form the remedy is administered. If under such circumstances, the fever should reappear, it is no longer possible to cure it. If large doses are repeated daily for a long period, there is manifested (besides the pains in the stomach), a peculiar kind of fever, described by Bretonneau, and which assumes an intermittent type when the cinchona is given in an intermittent manner. This fever forms a sort of vicious circle within which inexperienced physicians revolve: ignorant of the action of the cinchona, they redouble their doses, and throw the patient into a state which may be one of great gravity.

There is another inconvenience: it results from habit [*accoutumance*] if I may be allowed to employ that old term. The patients, from constantly taking cinchona, become at last insensible to its action, and the fever is renewed notwithstanding the daily administration of the remedy.

The methods of Sydenham, and Bretonneau, as well as my own method, are not liable to these inconveniences.

Though the accidents which I have now been pointing out to you may be imputed to cinchona, we cannot blame it for causing engorgement of the spleen and the consecutive dropsical affections, though, in the period immediately subsequent to the discovery of the Peruvian bark, these evil effects were attributed to it. Notwithstanding the renewal in our day of this old controversy, the matter is now definitively settled, it being agreed, that this valuable remedy has no part in producing the organic lesions in question, which are the result of a poisoned saturation of the system.

I am no believer, however, in the wonderful and sudden manifestations of the virtues of sulphate of quinine, which, according to some persons, reduces the volume of the spleen within a few minutes of its administration. I have shown you a young woman stricken by paludal fever contracted in Guadaloupe, in whom the spleen was of enormous size, and in whom the abdominal walls were so attenuated that the entire outline of the engorged viscus could be felt. This case presented a very valuable opportunity to try the sudden action on the spleen of the preparations of cinchona: it did not necessitate recourse to the artifices of percussion, which only deceive beginners. Now, in this woman, the spleen not only did not instantaneously diminish under the influence of the remedy, but *for the first twelve hours it increased in volume*, rapidly diminishing during the following days. You all had an opportunity of observing this fact.

By what channels is cinchona introduced into the system? Generally, it is administered by the mouth; but there are circumstances in which this mode has to be relinquished.

Some patients have an absolute repugnance to swallow cinchona, whatever means may be taken to mask its bitter taste: there are others, particularly young children, in whom it produces vomiting as soon as it is swallowed. Administration by the mouth is also ineligible in those cases in which a gastritis or a severe gastralgia has been induced by the long continued use of potions and powders containing preparations of cinchona. Then, again, in certain pernicious fevers—in which, as I shall immediately explain to you, it is important to act promptly—in certain pernicious fevers, in cardialgia, and in cholera, the vomiting characteristic of the disease makes it sometimes impossible to administer by the mouth the smallest dose of cinchona or sulphate of quinine.

Under such circumstances, it becomes necessary to introduce the remedy by some other way; and the rectum, affording the greatest facility, is the way chosen. The doses of cinchona given by enema ought to be a little smaller than those prescribed to be taken in potions, because absorption takes place more rapidly and better in the large intestine than in the stomach. Should it be found that the rectum retains the cinchona badly, the doses must be repeated in such a manner as to insure absorption of the requisite quantity. Should the intestine ultimately become too irritable, the *endermic method* must be employed. The simplest proceeding which can be recommended consists in employing poultices made with wine and powder of cinchona. These poultices ought to be very large, and must be kept applied to the abdomen for eight or ten hours. I should not be inclined to rely on their efficacy.

To render the cutaneous absorption of the remedy more active, M. Lembert has proposed to make the application to the skin after having removed the epidermis. Crude cinchona ought hardly ever to be employed in this way; but there is no objection to applying the sulphate of quinine to the denuded dermis; and it has been said, that by its application in this manner, intermittent fever may be cured with as much certainty as if the medicine were administered by the mouth or rectum. Certain precautions have to be taken. Dr. Briquet's experiments[1] show, that a solution of sulphate of quinine applied to a blistered surface only produces very slight pricking and a little local irritation, while in the state of powder, it occasions acute smarting, or pain more or less intense; and that if the powder be applied several days, in succession, it may act as a caustic causing the formation of a slough, and consecutive ulceration. This I mentioned long ago in my *Traité de Thérapeutique.*

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[1] BRIQUET:—Recherches Expérimentales sur les Propriétés de Quinquina et de ses Composés. 2me édition : 1855.

At the commencement of the treatment, particularly in the intermittent fevers of spring and autumn, I generally give an emetic, and often even a purgative, combined with cinchona according to the following formula:—

> Boil fifteen grammes of yellow cinchona bark in three hundred grammes of water, so as reduce the quantity to two hundred and fifty grammes: in this, dissolve twenty-five grammes of the sulphate of soda.

The patient is ordered to take this potion in two draughts, leaving an interval of half an hour between them.

I have now, Gentlemen, explained at great length the regular method of treating legitimate intermittent fevers; and it now remains for me to tell you how to combat *pernicious fevers.*

We are indebted to Morton for having formulated the beneficial effect of cinchona in these fevers. He did not, however, point out the method by which we may almost always triumph; and Torti was the first to lay down sure rules for the treatment of these formidable diseases. Abandoning Morton's plan of giving four grammes of the cinchona every three or four hours (in every respect a faulty proceeding at least when we have to do with a pernicious quartan, which has a long apyrexial period), Torti shows, that in pernicious subintrants, or in those which are really remittents, it is necessary to gain time by giving the cinchona in doses thrice as large as those administered in simple intermittent fevers.

Consequently, he gave the patient, in one dose, from fifteen to twenty-four grammes of cinchona, taking care that the medicine was taken as long as possible before the next paroxysm. He did not give the remedy during the intermission (because there is seldom a complete intermission in pernicious fevers), but at the time when the previous paroxysm begins somewhat to subside—in a word at the beginning of the period of remission.

This method, though infinitely superior to that of Morton, is by no means faultless. It cannot be denied that in fevers which are tertian, pernicious, or subintrant, the interval between the remission from the preceding and the onset of the following paroxysm is often too short to allow the cinchona to be sufficiently absorbed to act usefully.

Bretonneau, impressed with the weight of this objection, modified Torti's method of treating pernicious fevers, in the same way that he had modified Torti's method of treating simple intermittent fevers. He prescribed that *the administration of the cinchona should commence the moment that the pernicious character of the fever was ascertained, even though it should be in the middle of a paroxysm.* By pursuing this plan, we can manage to obtain at least from twenty-four to thirty-six hours before the following attack begins, and so be able to prevent it. In thus giving the cinchona during the paroxysm, we must not be afraid of augmenting its intensity, for the medicine does not act till some hours after it has been administered, and, therefore, not till the remission is about to commence. Inasmuch as we have before us a space of time relatively pretty long, we are not obliged to give all at once so large a dose as that advised by Torti. Bretonneau recommends twelve grammes to be given at once, and the same quantity to be repeated every three hours till the patient has taken thirty-five grammes of the powder of cinchona.

This method is certainly superior to that of Torti; but while I adopt Torti's rule as to the time of administering the remedy, I think that here the sulphate of quinine is very much to be preferred to the powder of cinchona. In a disease, in fact, in which the most terrible accidents are imminent, in which it is often a question of life or death not to allow them to gain upon us, it is necessary, with all possible speed, to put the patient into a condition to sustain the shock and resist its effects. Now, as I have already stated, the cinchona yields its active principles too slowly, whereas the sulphate, particularly the bisulphate, of quinine are rapidly absorbed.

When you have to treat a case of pernicious intermittent fever, you must, therefore, administer to the patient, with the least possible delay, a large dose of the salt, say two or three grammes, in potion; or, if the stomach will not tolerate the potion, give it in enema: this dose must be repeated for five or six consecutive days, till the symptoms are arrested.

As soon as the danger is quite passed, it ceases to be necessary to give the remedy in such high doses. You then, resuming the medication suitable in simple intermittent fever, according to the method which I have pointed out, give cinchona in preference to sulphate of quinine, and continue it for one or two months—eight grammes every morning.

Simple *masked fevers*—neuralgic affections and neuroses masking marsh fever—demand a special treatment, in this sense, that the disease being out of its usual ways, is more difficult to suppress. Though the indications are the same in these cases as in ordinary intermittent fevers, in respect of the medication to be employed, that medication must be more active, and it is specially necessary to continue it for a longer period. Thus, the doses of cinchona or sulphate of quinine, which must be administered immediately after the paroxysm, require to be stronger than those employed to cut short a regular intermittent: and it will often be necessary to continue them for five or six days in succession so as to put an end to the symptoms. When they have been got rid of, it will still be requisite to persevere for a long time in the use of the same means, observing always the rules which I have laid down for your guidance.

Hitherto, I have only spoken of cinchona and the sulphate of quinine; but there are other substances derived from the bark, and one in particular, which merit special notice. The substances to which I refer are *cinchonine* and its salts, particularly the *sulphate of cinchonine:* they unquestionably possess, the febrifuge properties of the preparations of quinine, but in a very inferior degree. To attain the same results from their use, it is necessary to give them in doses twice as large. There is also *quinium,* or the alcoholic extract of cinchona (prepared by the lime process),[1] a medicine recently introduced into the materia medica by MM. Delondre and Labarraque, a substance which differs little from crude quinine, to which I must now briefly call your attention.

Crude quinine [*quinine brute*], as much a febrifuge as sulphate of quinine, has certain advantages over it, with which it is important that you should be made acquainted.

Its tastelessness renders it peculiarly valuable in the treatment of children, as it can with the greatest ease be administered without its being perceived, while it is impossible to disguise the bitterness of the sulphate of quinine. It is of resinous consistence, and softens with the heat of the fingers, in such a way that it can be reduced into the form of exceedingly minute pills, which can be easily swallowed in soup or preserves. Its dose is the same as that of sulphate of quinine.

I have not said anything to you regarding the *succedanea of cin-*

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[1] See the *Bulletin de l'Académie de Médecine.* Paris, 1857, T. xxii, p. 1008.

chona: nor should I now refer to the subject had I only to mention alleged specifics, such as salicine, olive, alkekenge, and sea-salt, for they do not merit detailed notice; but *arsenic* is a remedy which does not belong to this category. It was long ago employed in the treatment of intermittent fevers; and has been reinstated in its place of honour by my lamented confrère, Dr. Ch. M. Boudin,[1] whose successes have been confirmed by the successes of many other practitioners, who have been zealous in making them known through the medical press.

Such of you as wish for information regarding the history of this method of treatment, I refer to a chapter which Dr. Pidoux and I have dedicated to that subject in our treatise 'On Therapeutics.'[2] For the present, I shall confine myself to a statement of the rules which Dr. Boudin has laid down for the administration of his lauded remedy.

The following is the substance of his rules: to begin the treatment by prescribing an emetic consisting of one gramme of ipecacuan, and ten centigrammes of tartar emetic, with a view to combat the concomitant gastric disturbance, and the absence or diminution of appetite: then, to give the arsenious acid in frequent small doses, taking care that the last of them is administered two hours at least before the time at which the paroxysm is expected: to proportion the dose to the special character of the fever, which varies with the locality, the season, and the individual.

The dose is a milligramme, or half a milligramme, of arsenious acid, that is one gramme or half a gramme of the following solution:—

Arsenious acid	1 gramme.
Distilled water	1000 grammes.

As a precaution, it is indispensable to boil this solution for a quarter of an hour.

Fifty grammes of this solution represent five centigrammes of arsenious acid. The desired dose is mixed with an equal quantity of wine, infusion of coffee, or water.

This medicine may be administered equally well during the apyrexial day and the day of the paroxysm: the medication ought to be continued during a period proportionate to the time the disease has existed, and the greater or less degree in which it has resisted anterior treatment. In fevers of the first invasion, it ought to be continued for at least eight days after entire cessation of the paroxysms. In old and obstinate fevers, it is often necessary to prolong the use of the arsenious acid for thirty, forty, or fifty days, or even for a longer period.

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[1] BOUDIN: Traité des Fièvres Intermittentes, Rémittentes, et Continues des pays chauds et des contrées marécageuses, et de leur traitement par les préparations arsénicales. Paris: 1852.

[2] Second volume, p. 366 of the seventh edition.

The tolerance for the remedy is very variable in different individuals, and it likewise varies in the same person, diminishing for a time, and then becoming reestablished as before. A particular patient, who at the beginning of the treatment bore very well five centigrammes of arsenious acid, is unable to tolerate this dose two or three days afterwards, when the paroxysm is cut. The signs of this intolerance are nausea, headache, and impaired appetite; or the manifestations of intolerance may occur in a higher degree—in vomiting and diarrhœa.

It is necessary to follow attentively these oscillations of tolerance, so as to diminish doses with diminution of tolerance. It often happens that we are obliged to give the medicine by the rectum, by which mode of administration, the patient supports five, ten, or even twenty centigrammes of arsenious acid, when unable to bear one centigramme by the stomach.

From diversity of idiocyncrasy, it is very difficult to determine beforehand the doses which will be required to effect a cure. Dr. Boudin has often cut short a fever by one milligramme: in other cases, he has found it necessary to raise the dose to five centigrammes or more within the twenty-four hours.

One of the essential conditions of this treatment, is to keep the patient upon a diet as substantial and abundant as possible, the only limits being his appetite and digestive power.

You perceive that Dr. Boudin's treatment does not consist in substituting arsenic for cinchona, but in carrying out a complex medication, in which arsenic sets up an arsenical in opposition to the paludal diathesis, and is seconded by two powerful therapeutic agencies, viz., emetics given to combat the gastric disturbance and accelerate a restoration of appetite, and an alimentation which will shorten the period of convalescence, combat the tendency to relapses, and prevent many *consecutive accidents apparently connected with an impoverished state of the blood.*

In the treatment by cinchona, the accidents are combated by the cinchona, with which we alternate ferruginous preparations. In cases in which there is paludal cachexia, certain mineral waters, particularly those of Pougues, contribute usefully to the cure.

Under the influence of the arsenical treatment, as also under the influence of the other medications which I have brought under your notice—the preparations of iron, cinchona, and alcaline ferruginous waters—engorgements of the spleen, and other visceral engorgements disappear. Moreover, experience convinced Dr. Boudin that relapses occur much less frequently in individuals who have been treated by arsenic, than in those treated by sulphate of quinine. It remains to be ascertained whether the treatment by sulphate of quinine was carried out according to the good method which I have been teaching you, in the cases thus brought into comparison by Dr. Boudin.

Arsenic, like quinine, will be found an excellent prophylactic. As such, Dr. Boudin recommends it to be given in very small doses daily of about one milligramme.

Let me remind you, Gentlemen, in conclusion, that in recent times alcoholic liquors have been announced by Dr. J. Guyon as excellent succedanea for cinchona. I can give you no decided opinion as to the value of this kind of medication, which I have never had occasion to try. I may state, however, that one of my hospital colleagues, Dr. Hérard, physician to the Lariboisière hospital, tells me, that he has obtained wonderful results from it, particularly in a man suffering from African fever. A glass of pure rum, administered at the beginning of the paroxysm immediately subdued the shivering and cut short the symptoms. In a woman, however, who had contracted intermittent fever in one of the departments of France where such fevers are endemic, a similar experiment completely failed.

LECTURE LXXXVI.

RICKETS.

History.—Age at which Rickets usually shows itself.—General Appearance of the patient.—The disproportion between the size of the head and the smallness of the stature must not be confounded with what is seen in hydrocephalic persons.—Rachitic Deformities.—Order in which they occur.—Mechanism of their Production.—Fractures.—Anatomy and Physiological Pathology of Osseous Lesions.—Three Periods: period of fluxion and effusion: period of softening and transformation: period of reconstitution and consolidation.—A Fourth, consumption, may replace the third period.

GENTLEMEN:—Towards the middle of the 17th century, in the year 1630, the English mortuary tables, make mention, for the first time, of a disease, which the oldest inhabitants and the oldest practitioners of England did not remember to have seen prior to that date. It was called by non-professional persons "the rickets," a term probably derived from "*riquets,*" a word which in the Norman idiom of that period was applied to persons hump-backed or otherwise deformed.

Immediately afterwards, cases presented themselves in such numbers as to attract general attention. Several physicians who had had the best opportunity of studying them, met to communicate to one another the results of their observations; subsequently, three of the number, Glisson, Bate, and Regemorter were entrusted with the classification of the documents which had been collected. Afterwards, to give greater unity to the editing of the reports, Glisson undertook the task of writing the history of this singular disease, regarding which no trace could be found in the authors of the time, by the most attentive and erudite research. I cannot give the date of the first edition of Glisson's book; but I know that the second edition appeared in 1650. The work, which is written in Latin, is entitled "De Rachitide;" and *rachitis* is the translation into French of the name of the disease.

It matters little whether the name has or has not any claim to a scientific origin, whether Glisson, as has been said, struck with the deformity of the vertebral column in the majority of subjects, went to the Greek in search of his etymology—*νόσος τῆς ῥάχεως* (disease of the rachis) out of which he made *ῥαχέτις*, just as from *νόσος τῶν πλευρῶν* (disease of the pleuræ) is formed *πλευρέτις* pleurisy—or whether the term was adopted simply to recall the vulgar appellation, *rickets*. The name is good, and ought to be preserved; for, according to the judicious remark of Van Swieten, "*satis distinctum est abaliorum symptomatum et morborum nominibus, simulque satis facile pronunciatu est, nec difficulter memoria retinendum.*"

It has long been, and is now a question of discussion, whether this disease which all at once made so much noise in England was really as new as was supposed by Glisson and his fellow-workers. Van Swieten—who ought always to be consulted when we have to clear up an obscure point—adopts the views of the English physicians, devoting a long commentary to the controversy. A few years ago, Dr. Beylard, one of my old *chefs de clinique*, reviewing numerous works prior to his own, comes to an entirely different conclusion. Without at all questioning the merit which pertains to Glisson, he says that the disease was well known before that physician's day; and he goes back to Hippocrates and Galen (without whom it appears a history of rickets would be incomplete) for allusions to rickets. Then, skipping over ages, and coming down to the seventeenth century, Dr. Beylard cites Whistler, a German, as the author of a memoir on the same subject, and of earlier date than Glisson's work. It is true that this memoir was printed at Leyden in 1645, five years consequently, before the second edition of the English physician's treatise; but it is allowable to suppose that his first edition was published anterior to the memoir of the German and might even have been known to him.

Gentlemen, as you can understand, it is not easy to exhaust discussions of this kind. If rickets have always existed, it is astonishing that a disease so strikingly evident, and so well deserving of the attention of physicians, should not have been the subject of more detailed notices than those accidentally met with in the writings of past centuries. However, once the alert was sounded, rickets, or the *morbus anglicus,* as it was called on this side of the Channel, was soon pointed out in the different countries of Europe. In Germany, it was described under the name of *articuli duplicati :* in France, it was vulgarly called *chartre,* an old French word synonymous with *prison* (Lat. *castrum*), and which conveys the idea of the patients being imprisoned in an affection depriving them of the liberty of movement : it is also called *nouure des jointures:* speaking of rachitic subjects, they are said to be tied up [*noués*], an expression still current.

Whether rickets was or was not a new disease in the time of Glisson, whether he was or was not the first to speak of it, he had not the less the undoubted merit of having described it with such accuracy as to leave very little to be added to complete his picture.

Since Glisson wrote his work on rickets, numerous works on it have appeared. You will not ask me to give you a list of them. I shall only mention the authors of the more remarkable : among the older authors, are Duverney, J. L. Petit, and Levacher de la Feutrie: among the modern, are Rufz, Jules Guérin, and finally Dr. Beylard, who has, in his thesis, given the most complete analysis of the subject which we possess.[1]

Having established these preliminary points, I now proceed to the history of rickets examples of which occur in several children now in Saint-Bernard's ward.

Besides these living examples, I place before you plates taken from Dr. Beylard's thesis, which are faithful representatives of individuals almost all of whom I saw. I will also show you the skeleton of a young rachitic girl, who died in my wards of the Hôpital des Enfans, so that you may be able to form as exact a conception as possible of the effects produced by the disease, and that you may the more easily fix in your minds the views which I shall now endeavour to present to you.

Rickets is a disease of early infancy : it generally supervenes at the epoch of dentition, that is to say, towards the end of the first year of life, or during the first six months of the second year. All authors agree on this point; but all likewise concur in admitting, that, in exceptional cases, it shows itself both before and after the period now indicated, and that instances occur in which it is congenital. Without at present speaking of osteomalacia, the rickets of adults and aged persons, to which I shall devote some special paragraphs, the two cases, pictures of which I now show you, are examples of rickets supervening in the second period of childhood. Such cases are of very rare occurrence; for in 346 cases collected by Dr. Jules Guérin, he found only five in which the disease was developed in persons between five and twelve years of age. Make particular note, Gentlemen, that the only question is as to the epoch at which the disease declares itself; for when rickets is arrested in its progress after having acquired a certain intensity, the lesions which it has produced often remain and are incurable, continuing for life, so that the unfortunate sufferers retain horrible deformities, the marks of the malady from which they have been long free, just as a person who has had Pott's vertebral disease, will retain an irremediable gibbosity, although the caries which destroyed the vertebræ had been arrested for a long time, and the bones had even become partially restored.

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[1] Rufz :—Gazette Médicale de Paris : 1834.
Guerin (Jules) :—Mémoire sur le Rachitis : 1839.
Beylard :—Du Rachitis, de la Fragilité des Os, de l'Ostéomalacie. [Thèse de Paris : 1852.]

Upon looking at a young rachitic child, one is struck with its attitude, its physiognomy, and the disproportion between its stature and the size of its head.

While a child of the same age, whether in its cradle or in its mother's arms, sits up willingly, and likes to move about its little limbs, the behaviour of the rachitic child is very different. It always keeps the recumbent position, whether in its bed or whether it be held in the arms. Whenever an attempt is made to change this position, should it even be to give it the breast when it is very hungry, it utters plaintive cries, as if it dreaded the pains which the pressure of the hands was about to occasion, and of which the experience of the past is remembered. The child suffers; and is not pacified till gently and fittingly held in the nurse's arms. When one attempts to make it stand up, it complains even more, and avoids supporting itself on its legs, so great is its debility, and so exceedingly sensitive are all its members.

Were we to judge by stature, we should suppose it younger than it really is. In its huddled up state, at two years, it hardly looks more than six months old. The little boy, whose picture I now show you, is twelve years of age, and he is not one meter in stature or in other words, has not attained the height of a child of three years.

Along with the diminutive height, there is a head of abnormal bulk. The forehead projects. The coronal and parietal protuberances are prominent. The top of the cranium is flattened in consequence of the separation of the parietal from the temporal bones, and the retroversion of the occipital bone. The fontanelles are abnormally open. This fact—*the persistance of the fontanelles*—has, in my opinion, a great significance. When I find this condition in a child at or above two years of years, I look upon it as a feature characteristic of rickets, even though no other sign be present.

As you are aware, Gentlemen, at birth, the cranium is still soft, and its ossification very little advanced towards the arch, which presents membranous spaces—*the fontanelles*—which separate the pieces constituting the lateral and superior parts of the cranium. You are also aware, that these membranous spaces are the seat of an ossific process which proceeds more or less quickly, but which, in general, is terminated towards the end of the second year, the cranial vault being then completely closed.

When at birth, ossification has already taken place, and when, consequently, no fontanelles exist, or when the process of ossification is accomplished with more than usual rapidity, the fontanelles closing during the first months of extra-uterine life, the individuals sometimes remain microcephalous, and this microcephalism is coincident with idiocy. The impediment presented to the free evolution of the encephalon emprisoned within its unyielding osseous walls, explains, to a certain extent, the arrested development of the intelligence and of all the cerebral functions. When, on the contrary, the fontanelles are late in closing, and the brain can, consequently, become more easily developed, there is greater development of the faculties of which that organ is the seat.

Dr. H. Roger has shown that ossification of the fontanelles takes place between the ages of fifteen months and three years and a half; that is to say, that it is exceedingly rare for it to be seen at fifteen months, and that it is always seen at three and a half years. In a normal state, the occlusion of the anterior fontanelle is completed between the ages of two and three. Rickets, and hydrocephalus retard or hinder this ossification—the one by impeding the deposit of osseous molecules in the fibro-cartilaginous tissues, the other by exerting an excentric action on the cranium, the sutures of which are thereby separated.[1]

The same accomplished physician has shown, that of all the affections of childhood rickets is that in which the blowing sound is most frequently heard over the sutures. In forty-seven rachitic children having open fontanelles, Dr. H. Roger noted the cephalic blowing in thirty, the blowing being intense. In ten cases in which it was absent, the rickets was either found in a slight degree, or did not exist. Consequently, says Dr. Roger—"from its great frequency, cephalic blowing may be regarded as a sign of rickets."[2] Previously, MM. Rilliet and Barthez, in accordance with Dr. Roger, and in opposition to the assertions of Dr. Fisher of Boston, had stated this blowing sound to be frequent in rickets, and rare in hydrocephalus: not being, however, always wanting in the latter, Dr. Roger remarks, that its absence or presence cannot be looked upon as an absolute differential sign.

Gentlemen, you must beware of mistaking that which occurs in rachitic, with that which occurs in hydrocephalic subjects in whom the head has a size disproportionately large to the rest of the body. In hydrocephalic subjects, on the contrary, the anomalous size of the cranium is coincident with atrophy, and not with development, of the brain. The intra-ventricular serous effusion, the cause of the increased dimensions of the head, before producing separation of the osseous parietes and enlargement of the fontanelles, has begun to compress, from within outwards, the cerebral mass, effacing the convolutions in such a way, that when the effused fluid has been evacuated, microcephalism is found.

These are very important distinctions, because they explain the differences in development of the intellectual faculties in hydrocephalic and rachitic individuals. Whilst the former—*hebetes et obliviosi*—present all the characters of idiocy, the rachitic, these little suffering creatures who are unable to move without assistance, generally possess a greater intelligence than other children of the same age:—*præmatura ingenii, acumine et sensuum sincero exercitio a coœtanis distinguuntur.*

Their physiognomy, so often stamped with sadness and suffering, their expression of countenance, their way of speaking, all denote an advanced development of the intellectual faculties.

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[1] Roger:—*Bulletin de la Société Médicale des Hôpitaux:* T. IV, 1860.

[2] Roger:—Recherches Cliniques sur l'Auscultation de la Tête: 1860.

But that which still more imparts to the physiognomy a peculiar stamp, that which makes the rachitic subject appear older than the small stature indicates, older indeed than the real age, is the conformation of the maxillæ, which, like the rest of the skeleton, are subject to the evil influence of the malady, which produces a great effect on the process of dentition.

I have already pointed out to you in a previous lecture[1] the influence of rickets on dentition: I must now revert to that subject.

When dentition has not commenced before the disclosure of the malady—which is very unusual—the evolution of the teeth is almost indefinitely retarded, and when it has commenced, it is interrupted. When the teeth have come forth, they become carious and black, as was pointed out by Glisson; they likewise become loose, and are ejected from their sockets.

This statement you can verify in the cases of the children now under your observation in the wards.

One of them, a year old, has not as yet got one tooth: another, sixteen months old, has only two: a third, twenty months old has only eight, though rachitic in a slight degree and late in becoming rachitic, not being affected with the malady till less than four months ago. If you had an opportunity of observing the future of these children, you would see their teeth fall out ere they attained three or four years of age.

I have stated the general rule; but there are cases in which the teeth remain till the period of the second dentition.

I consider this arrest in the progress of dentition as a phenomenon of so much importance, that when an infant of a year old suffers from teething without any teeth appearing, I suspect that rickets is threatened.

When the disease has attained a certain degree of advancement, we find, on stripping the patient, that there is *deformity of the trunk and limbs*.

The *chest* presents a very extraordinary conformation. Flattened laterally below the axillae, it projects anteriorly, presenting in front the appearance of the keel of a vessel, the patient being, as is vulgarly said, chicken-breasted: *instar carinæ navis aut pectoris gallinæ*. In place of being convex throughout, the chest is convex in front, but sunk in and hollow at the sides: if you apply a straight ruler to the lateral parietes, and let it fall from a perpendicular line to the bottom of the concavity, it will be found to measure three, four, or five centimeters in depth. At the junction of the ribs with the sternal cartilages, there are small nodulated projections, which, from their globular form and mode of arrangement, have been compared to the beads of a chaplet. This is what has been termed the rachitic chaplet, the knots of which are formed by swellings of the sternal extremities of the ribs. The appearance of this chaplet is one of the earliest phenomena of rickets.

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[1] VOLUME IV: p. 157.

The flattening of the chest, and its consequent contraction, are only conspicuous from the third to the ninth rib. Although the upper part of the chest may seem to be equally contracted, in reality it has not lost capacity in that situation, as is easily perceived by looking at the skeleton of a rachitic person. The appearance which is so deceitful in the living subject, arises from the shoulders being approximated to one another by shortening of the *clavicules*, which bones instead of being like an elongated letter S (as in the healthy subject) are like a flattened S.

From the ninth or tenth rib, the thoracic cage at once enlarges, so as to resemble an inverted pelvis in which are lodged the abdominal viscera.

Posteriorly, the natural concavity of the cervical region of the vertebral column is exceedingly increased, while the convexity of the dorsal region makes an anomalous projection—a gibbosity of a more or less decided character.

This deformity, this diminution in the capacity of the chest, produces, as you may suppose, embarrassed respiration, and by a mechanism which it is interesting to study.

During inspiration, the diaphragm descends, and the ribs rise, so as to produce a tendency to a vacuum in the cavity of the chest. But at the same time, the inspired air passing through the trachea and bronchial tubes into the pulmonary cells, distends them to such a degree that the lungs are constantly in contact with the costal walls. There is, therefore, pressure exerted from within outwards by the inspired air, but pressure, insufficient to bring to an equilibrium the opposed pressure of the atmosphere: in the normal state, the want of equilibrium is compensated for by the resistance to the external atmospheric pressure presented by the osseous and muscular cage in which the lungs are enclosed. The history of penetrating wounds of the chest shows us what happens when the external air can enter the pleural cavity through an opening in the parietes of the chest: the lung is squeezed up on itself, the contained air being unable to contend against that which is pressing upon the outer surface of the lung. Suppose that the unperforated thoracic wall does not offer a sufficient resistance, that which happens in rickets will happen. The pressure of the atmosphere acting with equal energy on all parts of the chest, but posteriorly, the ribs, whilst they are supported by the vertebral column, resisting this pressure, and anteriorly, where they are supported by the sternum, the ribs, soft and flexible, bend inward at their middle. Hence results that subaxillary flattening of which I have just been speaking, whilst the sternum is projected forwards, and the dorsal portion of the vertebral column, projected backwards, forms that gibbosity with a very long diameter observed in the rachitic, a gibbosity very different from that met with in Pott's disease. It follows, at the same time, that while that transverse diameter of the thorax diminishes, its antero-posterior diameter increases.

Though the flattening is limited to the lateral parts of the thorax, while the upper part retains nearly its normal aptitude, and the lower part is dilated, it is at that upper part, the ribs, particularly the first, are stronger than those which follow: this arises, in the first place, from their being more protected in that situation than elsewhere by thick muscles, and secondly, from the clavicles being placed, like two buttresses, between the sternum and scapula.

Though the base of the chest is dilated, that arises from its being supported by the liver on one side and the spleen on the other, which viscera are themselves supported by the mass of the intestines, generally much developed in rachitic persons: the diaphragm also here plays its part. Gentlemen, you know that the beautiful electro-physiological experiments of Dr. Duchenne (de Boulogne)[1] have explained the action of the diaphragm in respiration. During the inspiratory movements, it rests on the organs contained in the abdomen, and in contracting, it raises and throws outwards the lower ribs upon those to which it is attached. Now, from the time when the superior costal respiration becomes embarrassed by the contraction of the chest towards its middle, the increased diaphragmatic breathing becomes supplementary: from an abnormal increase in the frequency of the movements of the diaphragm, there results a dilatation—a persistent dilatation—of the base of the chest.

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[1] Duchenne:—De l'Electrisation Localisée et de son application à la pathologie et à la thérapeutique: 2me édition: Paris, 1861.

This abdominal respiration is a phenomenon to which I wish to call your special attention. Should you observe it in a child otherwise well, and free from fever, the probable existence of rickets ought to suggest itself to your minds.

The embarrassment of the respiratory functions, which leads to embarrassment of the circulation (particularly of the venous circulation), explains to us the *enlargement of the subcutaneous veins*, particularly those of the head, which Glisson pointed out as usually occurring in rachitic persons.

The embarrassment in the circulation perhaps also explains the *profuse sweating*, so common an epiphenomenon in the disease of which I am now speaking.

The base of the chest is confounded in appearance with the abdomen, the volume of which is much greater than natural in individuals of the same age: this abdominal enlargement often misleads not only the family of the patient, but also inexperienced physicians, leading them to believe in the existence of the affection known under the name of tabes mesenterica [*carreau*], constituted by tuberculous adenitis of the mesentery, or tuberculous peritonitis, both of which affections are essentially different from rickets.

On palpation of the abdominal viscera, we find that the liver and spleen pass far beyond the margin of the false ribs under which they lie. There is increased resonance, on percussion, over the whole abdomen, due to great flatulent distension of the intestines. Sometimes, however, there is dulness in the lower and lateral parts of the abdomen: this dulness occurs along with fluctuation, and reveals a certain amount of effusion into the peritoneum. Cases of this kind are rare; and a great amount of ascitis is never met with. This is a fact which was noticed by Glisson, by whom none of the symptoms of the disease seem to have been overlooked.

The liver and spleen are not enlarged, though they pass beyond the margins of the false ribs. Their prominence in the abdominal cavity depends upon their being pushed down by the contraction of the thoracic cage. It is, therefore, a mistake to believe that there is hypertrophy of the liver in rickets: nevertheless, this erroneous belief is so common that large-livered (*enfant au gros foie—magno-hépate*) is a synonymous term with a rachitic child. I myself long participated in this error, and, twenty years ago, when I published my views on rickets, deceived by appearances, I inscribed hypertrophy of the liver and spleen among common incidents of the disease. Later researches, and more minute study, convinced me that this hypertrophy was, on the contrary, an exceptional occurrence, and that the organs in question were only displaced in rickets.

The increased bulk of the belly in rachitic subjects is therefore due to the pushing down of the viscera contained in the abdomen—which viscera in a normal state are concealed under the ribs. It is due to that cause, but, also, even more, to the gaseous distension of the intestines: it also arises from the digestive apparatus itself being much more developed in these young persons than is natural in subjects of similar age, a fact which Glisson explains by rachitic persons being usually very large eaters, the same result taking place in them as in those animals which eat daily a great bulk of food.

This voluminous belly has as its base a pelvis which has undergone modifications, the development of which, as well as of the abdominal viscera, it partly explains. Thus, while the cavity of the true pelvis is contracted, the large pelvis has, on the contrary, a greater capacity. The bones which enter into its formation being flattened and turned outwards, the iliac fossæ are consequently widened. This depends upon softening of the bones of the pelvis, in common with all the bones of the body, as part of the disease. The iliac bones yield to the pressure of the intestines, which, although they do not press strongly, exert a continuous pressure on these bones, which is augmented almost every moment by the respiratory movements. In this way, the greater pelvis becomes spread out, and this spreading out contributes to the contraction of the lesser pelvis, the ischia being approximated by the see-saw motion which gives them an inward bias; while, at the same time, the iliac portion of the greater pelvis is pushed outwards. This approximation of the ischia, which is promoted by the pressure of the femurs, leads to a change in the relative position of the coxo-femoral articulations: the cotyloidal cavities, which necessarily follow the ischia, approximate in the median line, their openings being directed downwards and a little inwards, in place of outwards and forwards as in their normal state.

The anomalous development of the abdomen, coincident with narrowing of the upper part of the chest, and a great increase in the size of the head, give the rachitic body a gourd-like form, a sort of figure **8** shape, if you will allow me to make such a comparison. To this deformed body are attached *members* the deformities of which are not less remarkable. Of course, I am now speaking of cases of rickets in which the disease has considerably advanced.

The thighs, separated from one another, form two arches, their concavity being directed inwards and a little backwards, the convexity being outwards and forwards. The legs have also lost their natural straightness. But this deformity almost invariably present in the thighs, where it is an exaggeration of the natural curve of the femurs, varies in the legs, not only in different individuals, but even in the same individual. In general, for example, before a child has begun to walk, the concavity of the curvature of the legs is directed forwards and inwards, in such a way, that the knees are widely separated from one another, and that the lower extremities, curved in their whole length, form, as the saying is, a parenthesis. When the children have walked, the deformity is constituted in the reverse manner, the concavity of the tibia and fibula being directed from without backwards, so that the knees approximate and the individuals are crook-kneed. In other cases, one of the two legs is bent outwards, and the other inwards, so that they fit into each other, so to speak; or they describe angles, arches, and Z shapes.

The same occurs in the upper extremities, with this difference, that the deformity is nearly always the same in the inferior, and varies much in the superior segment, the opposite of that which occurs in the lower extremities, as those just pointed out. Thus, almost invariably, the curved forearms have their concavity directed towards the palmar aspect of the hand, their convexity corresponding with the dorsal surface, whilst, although the arm is usually curved in the same direction, that is to say, concave forwards and inwards, convex backwards and outwards, it is not uncommon to observe an opposite arrangement, the humerus having its concavity backwards and outwards.

As I have already said, and as we shall see by and by, these deformities are due to softening of the bones: so great is the softening, that when one tries to bend the limbs in their continuity, very great flexion is the result. We must take care in this bending of the bones, to use great caution, otherwise we may produce fractures.

The articulations have undergone great changes. In the wrists and ancles, for instance, where these alterations are more manifest than elsewhere, there is visible at a glance, a considerable swelling of the articular extremities of the bones. It seems as if at the joints, a little above or a little below them, the limbs have been strongly bound round, so as to place them in the condition of trees the trunk and branches of which are strangled in such a way as to cause the formation of knots. This articular swelling is so constant, and at the same time so significant a condition, that the disease derives its vulgar name—*nouure*—from this peculiarity. This condition, which often appears at the commencement of rickets, prior to any other deformity, is the result both of this swelling of the ends of the bones and of the relaxation of the ligaments, the latter being sometimes so great as to permit the articular surfaces to separate from one another under the influence of even moderate traction, and that on fixing, for example, the fore-arm or leg, one can bend the hand or foot so as to cause them to describe a semicircle on their axis, the palmar aspect of the hand being brought into a line with the ulnar margin of the fore-arm, and to turn the foot from one side to another, the dorsal aspect being directed either inwards or outwards.

The relaxation of the ligaments which exists alike in the vertebral articulations, in the sacro-lumbar and sacro-pelvic articulations, combined with the softening and extreme sensibility of the bones, explains why rachitic children are so long in being able to walk, and why they are obliged to maintain the recumbent position.

Such, Gentlemen, is a picture of the characteristic deformities of rickets, a picture which you will find has an infinity of varieties: in some subjects, it will be as complete as I have presented it to you, and in others, some of the details which I have delineated will be wanting.

The deformities are the more striking, that they coexist with great emaciation. The muscles lack that firmness which they possess in healthy persons: both their bulk and power is diminished: *torositas musculorum minuitur ultra quam credi potest.* The cellular tissue which surrounds them is not filled with that hard thick fat which gives roundness to the form in healthy young children. The skin, shrivelled, flabby, and sallow, like a half-empty bag, covers the atrophied muscles, which form the only separation between it and the stunted, twisted bones.

Pursuing this subject still further, let us now study *the order of succession in which the deformities of the bones are produced:* let us endeavour to discover their *causes,* and explain their *mechanism.* We shall not, for the present, enter upon the pathological anatomy of rickets, but confine ourselves to what takes place in the living patient.

Dr. Jules Guérin, to whom science is indebted for remarkable and important researches connected with the subject now before us, has established the law, that rachitic deformities are always produced from below upwards, that is to say, that their first seat is in the lower extremities, that they appear first in the legs, then, in succession, gain the thighs, pelvis, vertebral column, and thorax, affecting last of all the upper extremities. So decided is his opinion on this subject, that he goes the length of asserting, that "all isolated deformities in the upper parts of the skeleton, in the vertebral column for example, when there is no deformity in the lower parts, are not due to rickets." This proposition is much too absolute. It is true, as a general statement, in respect of rickets in adults, although many exceptions occur: it is true, to a certain point, in respect of children who have begun to walk: but it is false, entirely false, when applied to very young children. In the latter, the deformities supervene in an inverse order to that indicated by Dr. Guérin. In them, they begin in the chest, trunk, and upper extremities.

These differences in the order of succession in which rachitic deformities occur, according to whether the individuals have or have not begun to walk at the epoch of the invasion of the disease, are explained by the causes themselves which produce them. These causes are pressure exerted continuously upon the softened bones, whether it be on the chest, in the manner I have explained to you, by the column of atmospheric air which surrounds it, or whether it be on the limbs or vertebral column by the weight which they have to support: this pressure, and the incessant action of the muscles inserted into the different points of the levers constituted by these bones, cause the deformities.

These causes acting together produce the double effect, at least upon the limbs, of producing a tendency to settling most marked in the short bones, a tendency to increase the natural curvatures specially marked in the long bones, and more decided in one direction than in another according to the disposition of the muscular apparatus in relation to the lever upon which this power acts, and according to the power and continuity of muscular action.

Let me explain myself. The muscular masses applied directly to the femur—for example, the biceps and crural triceps which envelope it, tend, by contracting in a line parallel to the great axis of that bone, to become shortened, to be huddled up on itself, when owing to the softening from which the patient has suffered, it no longer offers a sufficient resistance. On the other hand, the abductor muscles of the thigh, and those which flex the thigh on the pelvis, much more powerful than the abductors and extensors obliquely inserted at different points in the lower part of the thigh, tend to bend it, by approximating its upper and lower ends to one another, thus acting like the string of a bow. The result is an increase of the natural curve, which, in a normal state, is directed inwards and a little backwards. In the fore-arm, the mechanism is similar: the most powerful muscles, those situated on the anterior part of the member, and obliquely inserted in the lower parts of the radius and ulna, tend to bend these bones towards the palm of the hand, while the deep seated muscles, acting in a direction parallel to the axis of these bones, tend to huddle them together. Here, on the contrary, where, as in the legs and arms, the opposing muscles nearly antagonising one another, the deformities of the bones do not admit of being reduced to rule, and are completely subordinate, either to the pressure on the bones from without, or to the pressure exerted by the parts which they support.

This effect of the contraction of the muscles, which, combined with the effect of pressure, may be such as to break the bones, is very decided in the clavicles, which yield to the action of those muscles of the chest and back which are inserted in the clavicle and humerus and draw the shoulders towards the median line.

These, Gentlemen, are facts which to be understood, require only to be stated. You can also understand, without its being necessary for me to enlarge on the point, the manner in which pressure acts. I say nothing of what takes place in the chest, having already treated that part of the subject at sufficient length. I shall only allude to what takes place in the limbs and vertebral column: the latter is shortened from the squeezing up of the vertebræ, and is, at the same time, bent on itself in the direction of its natural curves, which, consequently, are increased.

You can understand from what I have said, why the osseous deformities are produced in so different an order of succession according to the age at which the individuals become influenced by the disease.

A child of fifteen or sixteen months is seized with rickets, some time after it has begun to walk. Its bones undergo progressive softening: those of the lower part of the body, which, when the individual is standing, support the entire weight of the body, are necessarily the first to become deformed. In these cases, the deformities take place in accordance with the law laid down by Dr. Guérin: the legs are affected in the first instance, then the thighs, pelvis, and vertebral column.

But in a child which has not yet walked, matters proceed differently. As it is always lying, its inferior extremities are not subjected to the pressure under which they yield in the other case. The only pressure which the body has to support is that of the surrounding atmosphere, a powerful and constant pressure which the thorax cannot resist, its osseous frame-work being softened. Hence it is, that in very young children, the first observed deformities characteristic of rickets are those of the chest.

Now comes the question:—How and why is it, that in a very young child, the upper extremities become deformed before the lower? The mother of one of our little patients has explained this to you:—"My child," she said, "cannot stand; and to enable him to sit down, he requires to rest on his hands." In this statement, you can at once perceive an answer to the question: we have, in fact, something very similar to that which I have just been telling you takes place in the walking child. The bones of the arm and fore-arm, which serve as a point of resistance, as the supporting basis of the whole of the upper part of the body, yield, in consequence of their softened state, under the weight they have to sustain.

I have been telling you, that under the pressure exerted on them, combined with muscular action, the bones of rachitic subjects may be broken: I may add, that an abrupt movement is sometimes sufficient to occasion such fractures. This is a not unfrequent complication of rickets. It is not unusual to find numerous fractures in the same patient—several bones broken, or the same bone broken in several places. When I go into details relative to the pathological anatomy of our subject, I shall have to examine the mechanism of these fractures. For the present, bear in mind, that they often remain unknown to the relations, and even to the physician. On the one hand, the slight cause which often produces them, the usual presence of pain in the limbs, and its small increase at the time of the accident, make its occurrence pass unperceived: on the other hand, when we do suspect the occurrence of the accident, the diagnosis remains very obscure, for a reason which I am now going to state. The deformities due to displacement of the fragments, when there is fracture, are difficult to distinguish from those which depend on increased curvature or deviations of the bones depending on rickets: generally, moreover, there is no displacement of fragments, the integrity of the thickened periosteum maintaining them in position, sometimes in so solid a manner as even to allow the limbs to be raised in one piece. From this point of view, rachitic fractures resemble false joints. Finally, crepitation, the sign of so much value in the diagnosis of fractures, fails us in rachitic fractures, from the softened surfaces in contact not being hard enough to produce crepitation.

We must not suppose, however, that the deformities of rickets are due exclusively to the action of the muscles, or to pressure, as they may be produced during intra-uterine life. The fœtus is very seldom affected with rickets: but cases are sometimes met with; and a very remarkable one has been seen recently by Dr. Peter at the Hospice des Enfans Assistés. The infant in question was eight days old when deposited at the hospital. It was rachitic in a very high degree: there existed great curvature of the upper and lower extremities, deformity of the thorax, and continued fever. But the most interesting feature in the case was the existence of callus, and indications of a consolidated fracture of the ulna, and of a consolidated fracture of the femur. The infant died on the second day after admission to the hospital. At the autopsy, Dr. Peter found the bones of the limbs in an abnormally flexible state, but not spongy. Callus was distinctly visible in the situations where its presence was diagnosed during life. This very interesting case not only proves the possibility of intra-uterine rickets, but it also demonstrates, Dr. Peter says, that the curvature of the limbs is produced in accordance with a law peculiar to the malady, rather than under the influence of weight or muscular contraction: and the existence of well-formed callus shows, that rachitic fractures may take place within the uterus, and that the state of rickets has long existed, as the fractures have had time to be produced, and time to be consolidated.

Dr. Peter failed to obtain any information from the relatives; and could not ascertain whether the precocious rickets depended on hereditary causes, or on other causes belonging to the individual's relationship.

Gentlemen, the study of the *pathological anatomy of rickets* is closely connected with the history of the deformities caused by the disease, and forms the complement of my remarks on its mechanism.

Our knowledge of this subject is of recent date. Glisson and the authors of last century were necessarily imperfectly informed on this part of the subject: they were quite aware that the softening of the bones constituted a leading characteristic of the disease: they also knew that there succeeded to this softening, at a given moment, consolidation of the bones which had been affected by it: but that was the extent of their knowledge. It is true that Duverney in his 'Treatise on Diseases of the Bones,' published in 1751, had entered into some details regarding the state of the bones in rachitic subjects. He had noted their rarefaction, their greater lightness, the roughness of their surfaces, due to the presence of layers of osseous matter *formed by the extravasation of the nutritive juices.* So entirely had these summary indications been forgotten, that, in reality, our knowledge of the subject dates only from our own day.

The first researches undertaken in this direction were those published in 1834 by Dr. Rufz.[1] Dr. Jules Guérin's long and substantial memoir appeared in 1839.[2]

A first period—*a period of incubation or effusion*—is characterised by a sanguinolent effusion into all the interstices of the osseous system, which seems as if infiltrated with blood less viscid and less consistent than that contained in the blood-vessels.

A second period—*a period of deformity*—is characterised by the development of a very fine, spongy tissue in the epiphyses and interstices of the long bones, and also between the periosteum of the bone, which latter becomes soft and bent.

A third period—*a period of absorption and consolidation*—is characterised by the transformation of this spongy tissue into a compact tissue denser and closer than the normal tissue of the bone, a transformation which gives it an extraordinary degree of solidity, converting it into a true ivory.

To these three, Dr. Guérin adds a fourth period, which is observed in persons who have long suffered from the disease, and in whom the rachitic cachexia is developed: this is the period of *rachitic consumption.*

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[1] Rufz:—Recherches sur le Rachitisme chez les Enfants. [*Gazette Médicale de Paris,* 1834: *p.* 65.]

[2] Guérin (Jules):—Mémoire sur les Caractères Généraux du Rachitisme. [*Gazette Médicale de Paris,* 1839: *pp.* 443, 449, 481.]

I accept these divisions, I only change some of Dr. Guérin's terms, substituting for them others which seem to me to be more in harmony with the conditions they represent. I admit, then, a first period of *fluxion and effusion,* a second of *softening and transformation,* a third of *reconstitution and consolidation,* and a fourth of *consumption.* These periods I shall now describe to you.

In the *first period,* the bones are tumefied; and this tumefaction is specially very manifest in epiphyses of the long bones, in the short bones (with which the epiphyses have a great analogy), in the flat bones, of which the external and internal tables are attenuated, their diplöe being as if inflated.

The bodies of the long bones generally retain their curves and natural shapes; because the softening which the osseous tissue undergoes, and which advances to its highest degree in the succeeding period, is not yet sufficiently advanced to yield to the influences of pressure and muscular contraction, the active causes, as I have told you, of rachitic deformities.

This softening, however, is now sufficient to deprive the bones of their consistence, and impart to them a certain amount of elasticity. If we make pressure with the finger, or only with the nail, on a point in the diaphysis of a long bone, we depress it, we crush it: if we squeeze somewhat vigorously a portion of an epiphysis, short bone, or flat bone, we readily cause flattening: if we endeavour to cut one of these bones with the scalpel, we succeed without any difficulty, and this we can do only in the skeleton of a person who has died of rickets.

On examining the incision, we see that the tumefaction is due to the areolæ of the spongy tissue of the short bones, and to the diplöe of the flat bones having experienced incipient dilatation, and partly to the concentric lamellæ forming the diaphyses of the long bones being more or less separated from one another. These areolæ and interlamellar spaces are filled with a fatty, gelatiniform, sanguinolent matter, which, in consistence and colour, resembles pale red currant jelly. This matter, which exudes from all the pores of the divided bone, a real matrix of new tissue which we see developed at a later period, likewise fills the medullary canal of the long bones, and is found interposed between their external surface and their periosteum.

The periosteum itself has undergone changes. More vascular than in the natural state, and injected with blood, it presents a bright pink colour: thicker than it usually is, it adheres intimately to the bone, which is very vascular and has lost its smoothness of surface.

The cartilaginous plate, which in young subjects separates the epiphyses from the diaphyses of the long bones, has a softened, bluish, and semitransparent tint, which disappears after some days of maceration in water; so that the epiphyses become completely detached from the bodies of the bones.

When dry, these tumefied inflated-like bones have lost density and weight: their spongy tissue is formed of cellules much larger than those which exist in healthy bone, some being enlarged, and others constituted by the union of several, which have had their walls destroyed or torn: their compact tissue is perforated by small holes, formed partly by interstitial absorption, and partly by disappearance of the vessels of the new formation which traversed them in the recent state.

In *the second period* of softening and transformation, the swelling of the epiphyses of the long bones is much increased, and towards the end of that period, it is also very great: in the short bones (of which the external plate of compact tissue becomes porous, whilst the areolæ of their spongy tissue enlarge), and in the flat bones (the attenuated external and internal tables of which are soft and elastic, and in a state to be indented by pressure with the fingers whilst their dilated diplöe is constituted by large cellules of a slightly resisting texture containing a violet coloured medullary juice, visible through the transparent laminæ of the compact tissue).

The diaphyses of the long bones swell out, or seem to do so, in consequence of a very peculiar disposition of the medullary membrane and still more of the periosteum, according as the normal direction remains or is modified by deformities.

Thus, the periosteum is much more vascular than in the first period, and the gelatiniform effusion between it and the body of the bone is also much greater: this infiltration of gelatiniform matter has assumed, moreover, enormous proportions wherever it has been effused. It is also transformed into a tissue which is reddish, but less red than the liquid from which it has been constituted. Very elastic, resembling a very fine sponge, this new spongy tissue is easily distinguishable from the old spongy tissue, by its fibres being more dense, more compact, and paler.

It is then that the bones have undergone a notable softening. On grasping them with the fingers, you will be able to bend them with wonderful facility. In the living rachitic subject, we have already found how easy it is to curve the limbs. It is at this period, that you can cut them across as you would slice a carrot, a root, or a soft branch; or, you can cut them into longitudinal strips, which may be bent and rebent without being broken.

There is, therefore, in this second period of the malady a very manifest softening of the old tissue of the bone, the lamellæ of which have become less numerous, more detached from one another, and more pliable than in the preceding period: at the same time, the formation of a new bony tissue constituted by the gelatiniform matter which began to be effused in the first period, now augments in quantity, and assumes a more and more marked cartilaginous consistence. There is, in a word, disorganization of the old osseous tissue, in which the concentric lamellæ of the long bones, the plates constituting the spongy tissues of the epiphyses and of the short bones are still more separated from each other, and are also less complete than during the preceding period. So great is the destruction of the osseous lamellæ, that when the bones are dry, on injecting water into that which constitutes the compact tissue of a long bone, it is found to traverse the tissue from one end to another, gaining in succession the different layers; and on placing a short bone before the mouth, we find that we can breathe through it, so great is its porosity. But, simultaneously with this disorganization of the old osseous tissue, reconstitution of a new bony tissue is taking place.

When this process of reconstitution is very active, the entire bone seems to consist of the gelatinous matter of new formation, in which there is found the substance of the old tissue softened, and the most solid plates of which are those next the medullary membrane, which is very vascular and very thick, as is also the periosteum.

A minute ago, I was telling you that the modifications of the medullary membrane and the periosteum vary according to the presence or absence of deformity. I am now going to describe these modifications, which are coincident with those of the bone itself, and its medullary canal.

If you examine the diaphysis of a long bone which has nearly retained its normal straightness, its periosteum will present neither unusual density nor unusual thickness. Sometimes, the periosteum does not adhere closely to the surface which it covers, but at other times, it adheres to it so firmly, that an attempt to separate it, will tear out along with it, the most external layer of the bone. In that case, the external surface of the one, and the internal surface of the other, are rugose, covered with small osseous points, which communicate to the finger, when passed over it, the sensation produced by feeling a fine file.

If you examine an abnormally curved bone, you ascertain its different dispositions in the concavity and convexity of its curves. In the concavity, the periosteum, red, and greatly injected, is more or less thickened. It is also more adherent to the bone, from which often it cannot be detached without tearing away a part of the osseous tissue itself, or rather the newly formed tissue exuded upon its internal surface.

This new tissue, which in that situation is found in great quantity, looks like cartilage, or bone, softened by acid. Its most internal layers are constituted by the old tissue, the primitive layers of which are separated from one another by the effusion: its more external layers, those (which form the greater part of the whole) are evidently new products of periosteal secretion. The formative process is exactly similar to that by which the formation of callus takes place in fractures. The analogy is the more complete, that the new tissue is about to be transformed into an osseous tissue much more solid, and much more dense, than the old tissue of the bone: this change is absolutely similar to that which takes place in respect of callus.

In the convexity of the deformed bones, the periosteum has, on the contrary, lost its normal thickness, which is accounted for by the compression of, and friction upon, that part exerted by the surrounding muscles, and perhaps also by other causes of which I am ignorant.

The medullary canal filled, as I have said, by the matter, everywhere effused, has lost its normal calibre. This contraction produced by thickening of its internal membrane, and the effusion of matter into the canal, is greatest where the curvature of the bone is greatest. When the curvature exists in a high degree, the medullary canal terminates abruptly at the convexity, and opens under the periosteum, meeting, at an obtuse angle, the other portion from which it has been separated by the new tissue formed in the concavity of the curvature.

Let us now see, Gentlemen, what takes place in *rachitic fractures.*

You can obtain a very complete idea of the facts which I am going to explain to you, by a glance at the preparations now before you: they present numerous examples of the different kinds of rachitic fractures.

These two femurs, both broken in two places, show you the fragments dovetailing, so to speak, the superior and inferior fragments respectively penetrating each other. These fractures are not consolidated; and this is the condition which you will find most frequently in the second period of rickets, when the fractures really give rise to false joints, that is to say, to a state of the parts similar to that which supervenes in ordinary fractures, in which the consolidation has been prevented by one cause or another, and in which the fragments remain united by a sort of elastic fibrous tissue. This fibro-elastic tissue is formed by the products of exudation furnished by the medullary membrane and the periosteum; and in addition, in rickets, by the cartilaginiform matter, about which I have said so much, and which begins to be transformed, I repeat, in the second period.

It is especially in this second period of rickets, that the different parts of the skeleton have in the recent, and still more in the dry state, a very low specific gravity. Here you see on this table, the entire skeleton of a child of eight years of age, which hardly weighs one kilogramme, though it ought to weigh seven or eight. Observe how spongy the bones are: observe, that when they fall, they neither make any noise, nor rebound like normal bones. The tissue of the epiphyses, and of the short bones, and flat bones, are exceedingly rarefied. These puffed out bones resemble bits of sponge-cake, or bread made of gluten. The bones of the cranium are transparent from attenuation of their external table, which is riddled with holes.

In *the third period*, the period of reconstitution and consolidation, a change takes place somewhat analogous to that which takes place in the first period. In the second period, we have seen that the broken bones become the seat of a process similar to the reparative process which takes place in every fracture—fluxion of the periosteum and medullary membrane, effusion of the matter destined to become the matrix of the new tissue—a fluxion and effusion which are particularly manifest in the periosteum. The analogy, which I have already pointed out, and to which I again call your attention, between the process of consolidation in ordinary fractures, and reconstitution in rachitic bones, takes place in the third period of rickets.

In the midst of the fatty gelatiniform matter effused during the first period, which began to be organised, and had assumed the gelatiniform aspect in the second, we now see the development of calcareous, osseous centres—the first rudiments of the new bone which is being formed. In the long bones, it is in the walls of their diaphyses that we must seek for the most remarkable changes tending to the reconstitution of tissue. "The compact dovetailed layers, become," says Dr. Beylard, "thicker and denser: the new tissue which was deposited in the concentric spaces between the cylinders likewise acquires some consistence: and it is remarked, that between the small osseous bridges which they send forth by constituting cellules of different dimensions, the organization of the effused matter is accomplished by a layer of phosphate of lime finer than that in the normal state. This transformation is accomplished rather slowly when the lamellæ have undergone great separation. It is not till a much later stage that the walls of the whole diaphysis are found to be transformed into the compact homogeneous substance which acquires the hardness of ivory." Consolidation thus is accomplished by a kind of eburnation exactly like that which is formed by the callus of fractures. This, at least, is what occurs in respect of the fractures of long bones.

In the epiphyses, short bones, and flat bones, consolidation is not produced at the charge of the periosteum, but solely by the amorphous matter effused into the areolæ of the spongy tissue of these bones. In the epiphyseal extremities of the bone, a change takes place of an opposite kind from that which occurs in the diaphysis. Although in this situation the new tissue acquires the density of ivory, eburnation which is specially distinct at the places where the bones are fractured, and in the epiphyseal extremities, has a tendency to be partially absorbed, and to form areolæ in such a way as to suggest to the mind the appearance of normal spongy tissue.

This eburnation of rachitic bones is explained when one has seen that great vascularity at the period of softening and effusion, that extensive development of vessels, and that hyperæmic congestion which impart to the bone the appearance of inflamed bone. What takes place in rickets is, I repeat, analogous to the process of consolidation in fractures, and in cases of ostitis: thickening of the periosteum, inflammation, or, if the term *inflammation* do not state what actually takes place, let me say a pathological process, in virtue of which there is deposited in the cellular tissue, in a sort of matrix, materials destined either to form the solid callus, or to become new bone, callus, or new bone which will become harder than the old bone.

Sometimes the bones, which during the preceding periods presented greatly increased curvatures, commence, at that period of consolidation, to get so straight again that the deformities may at last almost wholly disappear.

Upon weighing the bones thus repaired, one is struck by seeing, that in some parts of the skeleton they have increased, and in others diminished in weight. The weight is increased, in those which have not undergone shortening, such as the bones of the cranium, which have augmented both in density and in thickness. As for the long bones: upon taking the comparative weights of, for example, the femur of a rachitic child and the femur of a non-rachitic child of the same age, you will find, that the former is much heavier in the mass than the latter; but you will also find that certain parts of the former—those which have acquired a greater density and become eburnated as it were—weigh, on the contrary, much more than parts of equal volume and length taken from the non-rachitic subject.

Such, Gentlemen, is the history of the ordinary osseous lesions which characterise rickets, when the disease is arrested, and the patient recovers. To complete the picture of the pathological anatomy, I have still to speak to you of that which supervenes in individuals who have fallen into a state of true rachitic cachexia.

In them, the period of consolidation gives place to *the period of consumption*. There is then no attempt at reconstitution of the osseous tissue: the bone remains rarefied: the matter effused into its areolæ, and into its interlamellar spaces, beneath the periosteum and medullary membrane, is not transformed into that cartilaginous tissue which ought itself to be transformed into new osseous tissue. The old osseous tissue is gradually absorbed.

In the recent state, the long bones are exceedingly soft, and also friable. They are reduced to a very thin shell, filled with fatty matter: in some places, they are whitish, or of a more or less red hue, and contain the débris of osseous plates.

In the dry state, these bones are extraordinarily light, friable, and brittle.

The lightness and friability are not less remarkable in the short than in the flat bones, the tables of which become excessively porous; whilst their diplöe, the cellules of which enlarge, assumes an aspect so much like that of paste which has risen, that I cannot make a more appropriate comparison than to say that it is like a macaroon.

I must necessarily rapidly pass on from these questions, the full discussion of which would lead me far beyond the limits of the instruction appropriate for this place. To fill up the outline which I have sketched, I recommend you to read the chapter which Dr. Beylard has devoted to this subject in his work, to which I have several times alluded.

General Symptoms of Rickets.—Pains.—Loss of Flesh: Muscular Atrophy.—Profuse Sweats.—Embarrassed Respiration.—Progress of Rickets.—Death is in general the result of Thoracic Complications.—Etiology of Rickets.—Influence of Bad Diet.—Rickets must not be confounded with Scrofula.—OSTEOMALACIA, *or Rickets in Adults.—Treatment of Rickets.*

GENTLEMEN:—I have explained to you the physical or organic symptoms of rickets, among which osseous deformities occupy the most important place. I now resume the study of the symptomatology of the disease, and proceed to discuss another order of phenomena, that is to say, functional disorders. They have so great a value, that they often are sufficient in themselves to characterise the disease now under our consideration. They are the *earliest symptoms,* and as they manifest themselves before any appearance of pathognomonic accidents, are often difficult to realise; and they remain unknown to physicians either inexperienced or unaware of the possibility of their occurring.

In the first rank of this class of symptoms, are disturbances of the intellect, or rather, *special modifications of the mental condition.* There is, a certain kind of sadness analogous to that observed in cerebral affections, or, still better to indicate its nature, analogous to that gloom which takes possession of children who are hatching —forgive the expression—who are hatching the malady we term cerebral fever. You will understand that this symptom can only have a relative value, and is not one to which too much importance is to be attached, as it is met with not only in rickets, not only in cerebral fever, but likewise in many other diseases which in their commencement, or in their period of incubation, lead to a state of painful discomfort telling much upon the extreme mental mobility of children.

This mental gloom of rachitic patients depends, according to all appearances, upon their exquisite sensibility in every part of the body, a sensibility which I have been anxious to point out to you, which shows itself by eliciting plaintive cries of pain when an attempt is made to raise up the young patient. The unfortunate little creature, who up till then was enchanted by the caresses lavished upon it, appears now to be afraid of them; even an approach to the bed on which it lies, made as if with the object of changing its position, causes its countenance to express anxiety and fear. This change in the child's character, this fear which it shows of having pain roused up by the pressure of a hand, this habitual stamp of sadness on its countenance differ from anything seen at the commencement of other serious maladies, particularly from the prodromata of cerebral fever. Indeed, in a child stricken by that cruel affection, we can still produce a transient cheerfulness, causing it for the moment to emerge from its habitual melancholy languor. In the rachitic child, this is impossible. The more we try to excite it, or induce it to move, the more will it manifest impatience. It is heedless as to the games of which, formerly, it was fond. This repugnance to the amusements of its age, this habitual sadness in a child, which, with an appetite increased rather than diminished, loses flesh visibly, which always has an acceleration of pulse coincident with profuse sweats, are symptoms, I say, which have a certain meaning; for the child does not cough, and presents no sign which can give rise to a suspicion of the existence of tubercular phthisis.

These phenomena, in proportion as the child begins to walk, become more and more evident; the fever, or at all events, the acceleration of pulse continues; the skin is constantly covered with profuse sweat, whether the patient be sleeping or waking, or whether more or less covered than usual. The excessive perspiration is greatest in the head, and it is on that account necessary to change the child's cap and pillow-case several times a day, so quickly do they become soaked.

MM. Marchand, Otto, Weber, and some others, are of opinion, that the bones of rachitic subjects contain lactic acid and lactates, through the agency of which the phosphate of lime of the bones may be dissolved, and then absorbed. Thus it is, that the inorganic or saline matter goes on decreasing, and the organic or gelatinous matter becomes predominant: the result is softness of the bones. It appears from analyses recently made by M. J. Drivon, a distinguished *interne* of the hospitals of Lyons, that in osteomalacia the proportion of inorganic matter fell from 64 to 41 in the compact, and to 18 in the spongy substance: and he also found, that bones of rachitic subjects contain lactates, under the influence of which the phosphate of lime may be dissolved, and then absorbed.[1] My pupil, Dr. Peter, placing these two analyses side by side with the great clinical fact, the profuse sweating in rachitic subjects, asked, whether they were not connected. According to this view, the important and perhaps fundamental fact in rickets is the excessive production of lactic acid: a part of this acid would be eliminated in the sweat, which is known to be rich in lactates, and another portion, not eliminated, would act on the inorganic constituents of the bone and soften them. The sweats would be an emunction, but an emunction insufficient for the lactic acid secreted too abundantly in consequence of a vice in nutrition, the nature of which has not yet been determined.

It is then that the *pains* assume a character as to which no one can entertain any doubt. Then it is, that if an attempt be made to raise up the child in its bed, or, still more, if an attempt be made to lay hold of it, whatever precautions be used, it shows, by its cries, the sufferings which it is enduring. These sufferings are sometimes so great, that some subjects refuse absolutely to allow themselves to be touched, the nurse being obliged, when she suckles, to bend over the infant to enable it to get the nipple into its mouth.

It is then also, that we begin to perceive that there is *embarrasment of respiration:* this habitual oppression is a constant phenomenon in confirmed rickets, particularly when it occurs during the first two years of life. At a later age, that is to say, in children of three years, and still more in those that are older, it is an exceptional phenomenon. In the very young infant, it exists in an extreme degree. If you bear in mind my remarks on the progress of rickets, you will see the reason of these differences. Bear in mind, that at a very young age, the disease first appears in the chest, which is the seat of the earliest deformities; while in children who have begun to walk, they are first seen in the lower extremities. Bear in mind the degree to which the thoracic deformities proceed, and the extent to which the play of the respiratory organs is embarrassed.

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[1] Drivon:—*L'Union Médicale,* for September, 1867.

At this period of rickets, the loss of flesh becomes extreme; and this has engaged your special attention at the bedside of the little patients in our wards: this emaciation goes on increasing up to the time at which the malady is arrested. It is not only emaciation, that is to say, disappearance of the subcutaneous and intermuscular fat, but also, as I have already told you, a true atrophy of the muscles, which in rickets proceeds to an extreme degree, reducing the muscles to mere small fibrinous bands.

Atrophy of the muscles, softness of the bones, and excessive sensibility of every part of the body are the causes of the modifications which supervene in the functions of locomotion: they are the causes of the laziness to move which the children show, and the repugnance they manifest to remain in any other than the horizontal position.

I am not afraid to return to facts to which I have already called your attention: their importance is an excuse for repetitions. A child, which, up to the invasion of the malady, could perfectly well stand upon its legs, when supported by the arms, or held round the waist, which with energy straightened itself, and threw about its feet on the bed or piece of furniture on which it was placed, no longer does anything of the kind. If taken out of its bed, it crouches up, draws its knees up to its belly bending the legs on the thighs, and the thighs on the pelvis, absolutely refusing to hold itself straight up. The child which used to sit perfectly well in its nurse's arms can do so no longer. Should it have begun to walk, its walking becomes slower, difficult, and unsteady; and after some weeks, it will drag itself painfully by the side of pieces of furniture which it uses as resisting points, and is afraid to relinquish: it will then become wholly unable to get up, and will, consequently, remain constantly in bed.

Let us now study what takes place in the great functions of organic life.

First of all, let us consider the *organs of digestion*: the appetite which at the commencement of the disease had retained its regularity, which may even have been notably increased, diminishes proportionably with the advance of the rickets, and is quite lost when the disease has reached its climax. The great secreting organs, the cutaneous and renal systems, are functionally disturbed. The skin is covered with *profuse sweats*, and as a consequence of this increased perspiration, sudoral eruptions supervene. The urine also is generally abundant, though cases occur in which it is scanty: it is always pale, and there is a deposit which chemical analysis shows to consist of calcareous phosphates.

This incessant waste, and the coincident continuous fever, explain the emaciation of the patients. Nutrition goes on the more badly, that, in addition to loss of appetite, the digestive functions are seriously disturbed. Obstinate constipation, alternating with a diarrhœa which is rebellious against all treatment, an intestinal flux, contribute to augment the general debility.

The embarrassment of the respiration, which plays so great a part in the disturbance of nutrition by rendering imperfect the process of hematosis, makes rachitic more liable than other children to acute pulmonary affections, bronchial catarrhs, catarrhal pneumonias, which, in consequence of the obstacle to free respiration occasioned by the thoracic deformity, assume a very great degree of gravity. Pulmonary tuberculosis is a very unusual complication of rickets: to this remarkable fact, I shall have to direct your very special attention.

This observation suggests the question:—How do the rachitic die? But another question has first to be answered:—*What is the progress of the disease?*

Under certain circumstances, the progress is rapid. You will see children reach the last period, the period of consumption, in three or four months. In other cases, the softening of the bones does not attain its maximum for a year, eighteen months, or perhaps not for two years. Generally, rickets runs its course in six, eight, ten, or twelve months. Then, after a period of two or three years, consolidation is effected, and the patient is cured, but considerable irremediable deformities remain.

If matters always had this issue, rickets could not be considered as a serious disease, at least not serious in the sense of danger to life. Unfortunately, however, a fatal issue is often caused by the accidents with which it becomes complicated.

Of these accidents, the most important are thoracic; and, in fact, it is by pulmonary affections that the majority of rachitic subjects are carried off. Others die exhausted by the profound disturbance of the functions of nutrition, digestion, hematosis, and of the cutaneous secretions, of which I have just been making a rapid review.

I now come, Gentlemen, to a great question—*the etiology of rickets*—a question of far greater importance than one might at first be inclined to suppose. Numerous causes of rickets have been enumerated; but I shall confine my remarks to those which are in my opinion of incontestable influence, believing that the mention of the others would be both tedious and fastidious.

Of unimportant alleged causes there is one, however, which deserves to be discussed.

It has been said, and the opinion has been held by the most eminent physicians, that the scrofulous constitution plays a most important part in the production of rickets: confounding the two diseases with one another, it has been alleged, that the difference between rickets and scrofula exists only in form, the latter being only a manifestation of the former, just as are the glandular congestions, and all the alterations of bone observed in scrofulous subjects.

This is an error against which I have long protested. Having been entrusted for fourteen years with the medical charge of a large number of children at the Hôpital Necker, and having been subsequently four years at the head of a children's service in the Hôpital des Enfants Malades, I have had occasion to see and follow out a large number of rachitic subjects. Observing, with the most scrupulous care, the numerous facts which have come under my notice during that long period, and always finding a very great disparity between rickets and scrofula, I have established (as moreover was well said before me by Dr. Rufz) that rickets and scrofula not only are not manifestations of the same diathesis, but that, as a general rule, the one excludes the other. This law (also expressed by Dr. Jules Guérin), is so absolute, that you will have, *à priori*, almost a certainty of not finding tuberculous or scrofulous affections in a rachitic subject, nor rickets in one who is scrofulous or tuberculous.

There are, as I know, exceptions to this general rule. Cases have been published of this exceptional kind, and I could myself cite examples. These exceptions did not escape Glisson, who likewise pointed out the same distinction which I have established between rickets and scrofula: the exceptions are exceedingly rare, and are too few to weaken the law.

Even in their external characters, the two diseases present notable differences. Recall to your recollection the picture of rickets which

I drew for you, and compare it with that of scrofula: the differences between the two will then strike you as forcibly as they have struck me.

The scrofulous, far from being, like the rachitic subject, smaller than another child of the same age, is often remarkable for being taller. Its limbs, solid and resisting, present no deformities except when white swellings (not unusual in them, it is true) have attacked the joints, or when caries of the bones has led to ulcerations and indelible cicatrices. Except in cases of white swelling, the articulations are not deficient in firmness, are as well knit as in healthy persons, and offer neither the nodules nor irregularities of surface which characterise rickets.

In taking into account the period of life at which scrofula and rickets appear, the parallel will not be less striking. The one, as I have told you, appears in the earliest epoch of infancy, towards the end of the first year, or in the course of the second; for you must remember that I am not speaking at present of osteomalacia, the rickets of adults and aged persons, while scrofula shows itself especially in the second epoch of infancy. Follow an hospital service where the patients are children between birth and two years of age, and it may be long ere you meet with a scrofulous patient, while a month will seldom pass without your seeing rachitic patients.

After death, the scrofulous will almost invariably present organic tuberculous lesions, although they have succumbed to diseases of the bones, abdominal affections, or thoracic affections: you will almost invariably find, if not pulmonary tubercles at least tuberculous bronchial glands, which, in young children, are the most frequent manifestation of the diathesis: seldom, very seldom, I repeat, will you find traces of tubercle in the rachitic, even in those carried off by chronic pulmonary affections.

I insist on these points, because the confusion against which I wish to put you on your guard is still too common. How many physicians, allowing themselves to be imposed upon by appearances, on looking at two children, the one having an anomalous rachitic curve, the other having a gibbosity produced by the vertebral malady of Pott, believe that both patients have the same disease. How often, when an infant with a protuberant abdomen is presented to them, do they immediately conclude that it has the tuberculous affection of the mesenteric glands and peritoneum known under the name of *carreau*.[1] It is important for you to be acquainted with *carreau*, which cannot be looked upon as a disease of early infancy. During my long service at the Hôpital Necker, I did not meet within more than four children under two years of age who were affected with it. It hardly ever attacks children before the age of four or five; and very often, it attacks adolescents of from eleven to fourteen years. Do not forget this fact; and when consulted regarding children with large abdomen, under two years of age, the idea that the case is probably one of rickets ought first to present itself.

In a word, Gentlemen, scrofula plays no part in the etiology of rickets. It is not one of the causes of which I have now to speak.

Climate has an undoubted influence upon the development of rickets. The disease is unquestionably much more common in damp cold countries than elsewhere. There can be no doubt that it is observed more frequently in Holland, in England, and in certain localities in France than in other regions of Europe. This remark is equally applicable to man and the lower animals. Veterinary practitioners and breeders of stock will tell you that certain animals if shut up in damp places become rachitic, even when they have good alimentation.

But of the causes of this disease, *insufficient aliment* is the most powerful.

In his first works, Dr. Jules Guérin adopted this idea, that insufficient nourishment (and by that expression, vulgar prejudice understood lacteal alimentation, a too prolonged lactation) occasioned rickets and scrofula. From his usual talent of observation, he was not slow in perceiving that in exact opposition to that opinion, the children which become rachitic are not those too long suckled, but, on the contrary, those which have been prematurely weaned. It is quite correct to say the disease appears under the influence of insufficient alimentation; but then a very different meaning must be attached to the term insufficient alimentation from that generally received.

By experiments on the lower animals, the question was completely elucidated. Dr. Guérin sought to determine by experiments on animals whether rickets could be produced at pleasure. He took a certain number of puppies of the same litter; and after allowing them for some time to suck their mother, he abruptly weaned one half of them, which he fed on raw flesh, a kind of nourishment which might at first seem the most suitable for these carnivorous animals. In a short time, however, the puppies, which had continued to take the maternal milk, became strong and vigorous, whilst those which had been weaned, and placed upon an apparently substantial diet, became sad, were subject to attacks of vomiting, then became deformed in the limbs, and at the end of four or five months presented all the symptoms of confirmed rickets. From these experiments we must conclude with Dr. Guérin, that rickets is in great part dependant upon disorder in the function of nutrition, which is again dependant upon faulty feeding. Now, an untimely, is a vicious, alimentation. To feed carnivorous animals with flesh before they have passed the sucking age is vicious feeding; and from experiments made on pigs, it has been found to be equally deleterious to feed herbivorous animals with vegetable aliment, when they ought still to be at their mother's breast.

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[1] Tabes mesenterica.

Similar results will follow similarly faulty feeding in the human subject. In children, as in the young of the lower mammalia, milk is the only aliment which is suitable: it is the only food to which the digestive organs are adapted; and the absence of teeth is itself sufficient proof of this fact. I entered into this question at sufficient length when I spoke to you on the subject of weaning, to prevent the necessity of my again expatiating upon it. In respect of rickets, attentive observation convinces me that it is most common in infants weaned before dentition is sufficiently advanced, and fed on pap, vegetables, and even meat, in place of a milk regimen which is better adapted to their digestive aptitudes.

From what I have now said to you regarding this vicious alimentation, regarding the influence of residence in unhealthy localities, in damp, and badly ventilated places, you will be able to understand why rickets is observed more frequently among the poor, than among the comfortable classes of society. This, Gentlemen, is not because the prejudices relative to weaning do not prevail as much among the rich as among the poor, but because, along with that evil influence, the latter are subjected to other influences even more baneful.

As there can be no doubt as to the causes of rickets, does it follow, that they exist in all cases? Do children, ill-fed, living in deplorably bad hygienical conditions, inevitably become rachitic, and are those who are well-fed and living under the best possible sanatory conditions exempt from the disease? Certainly, in a general manner, it is correct to say that this exemption exists; but there are exceptions to the rule; and it is important that I should point out to you the possibility of their occurrence.

You will see children who have been suckled by excellent nurses, and reared under the most favorable possible conditions, become victims to this disease; whilst some wretched infants, prematurely weaned, or who without ever having been suckled at all, who have lived on the most indigestible aliments, and been destitute of every solace demanded at their tender age, do not become rachitic. I repeat, however, that these exceptional cases are of very rare occurrence; but they are sufficiently numerous to show that, in addition to the causes I have here pointed out, there remains one still to be mentioned—one which dominates all the others—*individual predisposition.*

This predisposition is often hereditary. No one disputes the part which hereditary predisposition plays in the etiology of rickets: not that the offspring of rachitic parents are necessarily rachitic, but that it is a fact established by observation, that such offspring is more liable to rickets than others, because the disease more readily developes itself in them under the influence of immediately exciting causes; and because, in particular, that when once it is developed, it is much more difficult to arrest its progress.

Gentlemen, the influence of hereditary predisposition has always appeared to me to be much more manifest in OSTEOMALACIA, the rickets of adults, regarding which I propose now to say a few words.

Here, a preliminary question presents itself. Ought we to consider, that there is a similarity between osteomalacia and rickets? I say we ought. In my opinion, and in that of many other physicians, they are one and the same: the differences between them belong to the difference of the conditions in which the economy has been surprised. These differences have much less bearing upon the general symptoms than upon the local accidents, that is to say, upon the osseous deformities, their order of succession, and the greater rarity of fractures in the rickets of adults than in the rickets of children. For this, there is a physiological reason. Osteomalacia supervenes at a period when the component parts of the skeleton have attained their complete development; and the rickets of children supervenes when the process of ossification is in progress, the result of which is, that the calcareous matter constituting the solid part of the bones, not being secreted after being absorbed under the influence of the morbid action as (it is secreted at an early age), the bones reduced to their fibrous parts, yield more easily, without breaking, to deformities, just as occurs in bones softened by the action of an acid. As to the order of succession in which these deformities take place, the details into which I entered at the beginning of this lecture will enable you to understand why it is, that in osteomalacia, the inferior extremities are first affected, and why in rickets, the pelvis is the earliest seat of the disease.

You can also understand, that curvatures and osseous deviations will infinitely vary according to the intensity and stage of the disease, and according to the extent to which the patients have used their limbs, these deformities being subordinate to the energy and frequency of the muscular contractions which largely contribute to produce them.

In the genesis of osteomalacia, as well as in that of the rickets of children, we can trace the influence of bad hygienical conditions, influences, however, much less marked in the development of osteomalacia than in the development of rickets of children. The conditions which dominate in the rickets of adults, and which specially merit your attention, are peculiar to the individual.

At puberty, that is, at the period of life nearest to the period of childhood, the disease most frequently developes itself. You know the rapidity with which the body grows during the early years of life. Consider the child in the interval which elapses between birth and the end of the third year: it grows so rapidly during that period, that were its growth to continue in the same ratio during following years, it would attain gigantic stature. From the beginning of the fourth year, however, growth proceeds more slowly up to the age of puberty, when it takes a fresh start; and then, it is not unusual to see the individual grow five or six inches in twelve months: then, also, the skeleton undergoes a modification similar to that which takes place in early infancy. When, these conditions existing, under the influence of immediate causes which generally escape notice, osteomalacia supervenes, it presents the greatest analogies to the rickets of children—I say *analogies*, not similitudes.

After puberty, there are other circumstances which may favour the development of the disease. Thus, it is not uncommon to see women attacked with rickets after having had several children, or even, it may be, after a first confinement, the disease beginning immediately after delivery. This influence of pregnancy, particularly of repeated pregnancies, is mentioned by many authors; and Dr. Beylard says, in his thesis,[1] that in 36 rickety women whose cases he collected, 15 had had children, 5 had never been mothers, and in respect of the other 16, there was no statement as to whether they had or had not had offspring. Dr. Beylard notices as a remarkable fact, that several of these women had become pregnant for the first time about the age of thirty.

You are acquainted with the researches of Dr. Ducrest in relation to the modifications experienced by the entire organism, particularly by the osseous system, during gestation.[2] It appears from these researches, that the state of pregnancy causes a certain amount of softening of the bones: the secretion of the solid materials which enter into their composition undergoes disturbances and notable deviations. These disturbances are indicated by the appearance of kystein, a sort of scum which appears on the surface of the urine of pregnant women; and which, according to M. Gubler, is a layer of ammoniaco-magnesian phosphate, on which there grows a cryptogamic vegetation, when the urine is allowed to remain at rest: this phosphate is found in excess in the urine of rickety children. These abnormal states of the secretion of calcareous matter are characterised by the bones, particularly those of the cranium, frequently presenting a remarkable thickness, caused by deposits in the form of osseous stalactites called *osteophytes*, which have been also found by M. Follin on the bones of the pelvis. There exists, therefore, in pregnant women, a certain kind of rickets, of which osteomalacia may be considered as a more advanced stage.

It appears that in a woman about to give birth, there occurs something analogous to that which takes place in plants when they are going to flower and fructify. At that period, plants undergo remarkable changes: thus, for example, the root of beetroot contains a large amount of sugar till the plant is going to blossom and form seed, when the sugar disappears. Here, then, is a remarkable change in the organisation of that vegetable. In female animals at the time of rut, modifications of an equally remarkable nature are observed. Both in vegetables and animals, then, the great and important function of reproduction is announced by momentous phenomena. Woman forms no exception to this great law of nature.

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[1] BEYLARD:—Du Rachitis, de la Fragilité des Os, de l'Ostéomalacie. Paris; 1852.

[2] DUCREST:—*Archives Générales de Médecine:* 4me Série, T. IV.

All her organism is in movement, if I may use the expression: all her systems, the osseous system included, undergo more or less considerable modifications: the modifications, however, are transient, for as soon as pregnancy has terminated, everything returns to its former state. It happens, however, that when these modifications have proceeded to extremes, if one may so speak, a return to the normal state is less speedily accomplished; and, under the influence of particular causes, the movement continues. The modifications to which the mother has been subjected, no longer responding to their original intention, operate injuriously on her: the modalities, even those which are physiological and transient, become pathological, constituting morbid states of greater or less gravity, manifested by symptoms which become more and more characteristic.

The case of a patient whom I treated in the Hôpital Necker, in 1848, a woman, aged 48, named Rehbin (which Dr. Beylard has published at full length), may be given as a complete and typical example of osteomalacia.

This woman considered that the malady which induced her to seek aid at the hospital had begun at the time of her first pregnancy. Her pregnancy took place at the age of thirty-two, and immediately after marriage. At the same epoch, her health began to be seriously deteriorated. Previously, however, the patient (who had always lived under very bad hygienical conditions), had suffered from nervous symptoms, connected with chlorosis dating from her twelfth year.

The symptoms which supervened on her becoming pregnant, were attacks of vomiting (to which we must not attach too much importance) and a general debility, accompanied by wandering pains in the back of the neck, the shoulders, the loins, the pelvis, and the lower extremities. These pains increased up to the end of pregnancy, and were characterised by shootings through the pelvis and thighs. However, notwithstanding her debility, the patient continued to walk, and to go out, up to the eighth month of gestation, when œdema of the lower extremities obliged her to keep her room.

Labour, which was natural, lasted twelve hours: the infant was at the full term, and well-formed. The mother left her bed at the end of fifteen days; but was soon attacked by fever, which recurred two or three times in the twenty-four hours: this state of fever, characterised by slight attacks of shivering, followed by moistness of the skin, lasted the whole period of lactation, that is to say for seventeen months.

The pains became more intense, always specially seated in the sacrum, manifesting themselves, likewise, by continual shootings through every part of the body, from the face, cheek-bones, and jaws, down to the feet and hands. Even gentle pressure on the bones increased the pain.

Six months after her confinement, the patient experienced difficulty in walking; and began to stoop. It soon became impossible for her to go about her house, or attend to her child. She hardly ever left home: and at the date of her giving up suckling her infant, she could only painfully drag herself along by leaning on the walls and furniture of her room. If she left the house, she was obliged to have recourse to crutches.

After a fall on the street, she took to her bed, which she kept several months. No fracture, however, was discovered. The pains became more intense, and she lost the power of moving. In changing her position, she required to take the greatest precautions She remarked, that her knees and feet turned outwards.

The general health continued good, to this extent, that digestion was accomplished in a normal manner, and menstruation became re-established with its accustomed regularity.

Two years after the birth of her first child, the woman Rehbin became again pregnant. At the end of the third month, her sufferings recommenced; and from that time, her pains became constant and general, occupying all the articulations, and becoming intensified on the least movement. She was compelled to remain at absolute rest, either lying down, or seated, supported by pillows, in an arm-chair: she reached her full time. Labour, which was otherwise natural, lasted twenty-four hours, that is, twice as long as on the first occasion. For some months after her confinement, a certain amount of amelioration took place: she was able to walk with the aid of crutches, which it was found necessary to shorten. The organic functions were performed naturally: menstruation was re-established six weeks after delivery. The pains only remained, and they had become more frequent, occupying the whole of the osseous frame, particularly the right side, and in a still more special manner, the pelvis and continuity of the limbs. So they continued, without much increase in intensity, till about fifteen months had elapsed from the birth of the second child.

The patient then became pregnant for the third time. Tingling sensations and feelings of numbness, which were experienced in the limbs towards the end of the first pregnancy, and had manifested themselves from time to time, now lasted for several hours, so as to lead the patient to fear that she was falling into a state of paralysis. These sensations were stronger in the upper extremities than in the legs and feet. The limbs were likewise liable to painful contractions which had occurred from the beginning of the malady, and returned irregularly. Under these circumstances, the woman reached the full term of her third pregnancy: she was then thirty-eight years of age. Labour, as on the two former occasions terminated naturally; but this time it lasted seventy-two hours. The child, though strong and well-formed, died when two days old.

After that confinement, the disease became greatly aggravated: the pains were constant: they became intolerable with any movement or on the slightest pressure: to such an extent was this the case, that the patient could hardly bear the weight of her clothes or blankets. During the night, the pains were more severe than at any other time, when they extorted cries from the unhappy sufferer.

Her figure had been crooked for a long time; but from the date of this third confinement, the deformities became great.

The face was shortened: the cheek-bones had acquired increased prominence: the superior maxilla projected so much as not to correspond with the lower jaw: from this cause, and the loss of several teeth leaving a gap, mastication was difficult, particularly when the food offered resistance to chewing.

The head, regarded as a whole, was, however, not deformed; although the cranium was exceedingly sensitive.

The cervical region was shortened, in consequence of increase in its curve forwards, and the squeezing together of the vertebræ.

The thorax was flattened from before backwards, and shortened from above downwards, being inclined towards the pubes. The ribs were imbricated, lying over one another in such a way as almost wholly to efface the intercostal spaces.

The pelvis was flattened in the same way as the thorax, that is to say, in its antero-posterior diameter. The iliac bones were turned outwards in a very obvious manner.

The thighs were shortened and curved, the hollow of the curve being directed backwards and inwards.

Over this curvature, the integuments were hard, and formed very prominent folds.

The legs and upper extremities presented no change, although for a long time the patient could only half supinate the hand.

The malady grew worse; but, nevertheless, the functions of organic life were not disturbed: there was no loss of appetite, digestion was easily performed, and the bowels were regularly opened every day. Her diet was wholesome, being composed of flesh and vegetables, with wine as the usual beverage.

This state of matters had continued several years, when a slough over the sacrum complicated the case. The patient was then admitted to my wards in the Hôpital Necker. As I have already told you, that occurred in 1848. The malady had then existed sixteen years; and during the latter half of that period had made great progress.

I was at once struck with the deformities of which I have now given you a condensed account. I was struck with the patient's diminutive stature: she told us that her height was formerly $1^{m.}$ 78, but was now reduced to one meter. So great was the deformity of the pelvis, that when I wished to make a digital examination, with a view to determine its internal dimensions, I found it impossible to pass my finger into the vagina, and was only able to introduce a pretty large sound.

Well, Gentlemen! this woman recovered: she recovered under the influence of cod-liver oil, which I prescribed for her from the time she came into the hospital, and which she continued to take long after she returned to her own home.

You understand that this recovery took place as recoveries take place in rickets. I was fortunate enough to arrest the progress of the disease, so as to procure consolidation of the bones: but I had no ground for hoping that I could remedy the deformities, which were necessarily persistent. Dr. Beylard, however, who saw the patient in 1851, ascertained that she had slightly regained her stature: on measuring her, he found that she had regained 43 centimeters: at that date, her general health was excellent, and she had become quite plump: except menstruation, which had not recurred for two years, all her functions were performed with perfect regularity. She complained, however, of some wandering pains, and of shooting pains supervening on sudden atmospheric changes. Although this case is full of interest in all its aspects, I must refer you for farther details to Dr. Beylard's thesis.

In another case, which occurred in my private practice, I likewise saw rickets supervene, consequent to, but several years after, a first confinement. The lady was married at twenty, and was delivered in 1831 of the only child she ever had; and which, in the following year, when enjoying perfect health, was carried off by an attack of cholera of a few hours' duration. This event occasioned profound grief to the mother, which time was unable to moderate.

A year afterwards, she fell into bad health. She had attacks of menorrhagia, recurring at short intervals; and she complained of incessant pains in the loins and pelvis. These pains soon extended to the back; and in 1835, an obvious rachitic deformity was perceived, the spine being curved forwards. Some months later, the curvature was more marked, and was likewise lateral: one shoulder also projected more than the other. These deformities went on increasing: in 1840, five years after they had commenced, seven years after the beginning of her malady, and nine years, consequently, after her confinement, they were exceedingly conspicuous. The projection of the shoulder was great: the stature was much diminished: there was collapse of the thorax from above downwards, to such an extent that the ribs, as it were, overlapped one another. At this period, the pains were such as to disable the patient from walking.

Now, for the first time, the nature of the malady was recognised. Forthwith, she began to take cod-liver oil. The anticipated success was obtained from that medication. In two months, the pains were sufficiently mitigated to enable her to walk, and after two years of perseverance in the treatment, it being only temporarily discontinued from time to time, so as not to avoid irritation of the digestive canal, the cure was complete, with the exception of the irremediable deformities.

In June 1863, you may have seen, in bed 3, Saint-Bernard's ward, a very rare case of rickets, or acute osteomalacia in an old woman of seventy years of age, who came into the hospital with cough, dyspnœa, and high fever. This patient complained of suffering horribly in certain parts of the sternum, and throughout nearly the entire extent of the ribs. At first, I supposed that she was exaggerating her sufferings. She shrunk from the hand when any attempt was made to examine her by palpation or percussion. This was something quite unusual: the pain was as intense as in the most violent neuralgia; but it was not circumscribed as in neuralgia.

On the contrary, it was diffuse and bilateral, which is unusual, intercostal neuralgia being generally seated in the left side. Then, again, there was fever. On percussing the chest, with a view to discover whether there existed fulness indicative of possible pleuritic effusion, I thought I perceived slight crepitation, while, at the same time, the ribs seemed to bend under the plessimeter. To make myself quite sure on this point, I pressed somewhat strongly on the convexity of the ribs; and there was then no doubt that the bones bent and crepitated under pressure: very acute pain was at the same time excited. Light now began to dawn; and I thought it probable, notwithstanding the rarity of such an affection, that it was rachitic softening of the ribs. In that case, the pain ought to be most acute at the junction of the ribs with their cartilages: and it was so. In these situations, there was tumefaction. Pressure produced acute suffering, and at the same time enabled one to perceive an abnormal mobility: to a certain extent, the cartilage could be made to ride upon the rib. Pursuing my investigations, I also made out that the sternal pain was not uniformly distributed, but was located at the junction of the first and second pieces of the sternum, and at the union of the second piece with the xiphoid appendix.

I therefore found myself confronted with painful softening of the ribs, and sternum, and pain seated in the favorite seats of pain in rickets, that is to say, in the articulation and epiphyses. Could any doubt have remained, it would have disappeared on a more complete examination of the woman, for she presented a very strong gibbosity. Her vertebral column was curved in the form of the arc of a circle of great diameter; and was not bent in an acute angle as in Pott's disease. The articular apophyses of the ribs were bulky, and the long bones were slightly deformed. Finally, the patient stated that she had been rickety [*nouée*] in childhood.

She had dyspnœa, due in part to the pain ocasioned by the respiratory movements, which caused the softened ribs to ride on one another, and in part also, to a slight bronchitis from which she had suffered for some days. She suffered habitually from palpitation, due perhaps to displacement of the heart, which was not enlarged.

The thoracic pains, which had set in six weeks previously, commenced with febrile excitement. The fever was constant, tolerably moderate during the day, liable to exacerbations in the evening and at night. Profuse sweating occurred towards morning.

I placed the patient on a cod-liver oil regimen, and tried to subdue the fever by administering the tincture of digitalis and the alcolate of aconite.

Fourteen days after admission, that is to say, on the 7th July, she had violent shivering, and on the following day, were perceived dulness, crepitation, and an obscure blowing in the right axillary region. Next day, this poor woman succumbed under an advancing pneumonia.

At the autopsy, we found a very altered state of the ribs and sternum. There remained only one very thin plate of compact tissue; and the superabundant spongy tissue was gorged with blackish blood. So extensive was the softening of the ribs, that they could be bent in all directions, and cut by a knife with the greatest ease. The sternum was in a similar state of softening.

It is a remarkable fact, that the long bones of the limbs presented no similar alteration: therefore, notwithstanding this woman's advanced age, the rachitic disease had, as in the infant, begun in the chest.

The whole of the upper half of the right lung was hepatised; and all the bronchial tubes were inflamed.

This case, in my opinion, decisively demonstrates the identity of rickets and osteomalacia: the lesions were certainly those of rickets, but the patient's age was that at which osteomalacia is usually observed. Moreover, she died of inflammation of the lungs, as generally happens in the severe form of rickets, as if the impediment to the respiration, by causing passive congestion, produced inflammation of the pulmonary parenchyma.

Gentlemen, I am also strongly led to believe, that rickets and osteomalacia are the same disease, by the fact, that both are wonderfully combated by the same medication.

This medication may be considered as really heroic in the *treatment of rickets:* it consists in giving cod-liver oil, and, in a more general way, fish oil. Though employed from time immemorial by the people in England, in Holland, in Westphalia, and on the northern coasts of Germany, it was never, till recently, prescribed by scientific practitioners. At the commencement of this century, two physicians on the other side of the Rhine, Schenck and Fehr, published some very interesting observations in relation to its use; but the facts to which I refer remained unknown in France, till 1827, when Bretonneau, who, like every body else, was ignorant of them, was led, in the manner I am now going to relate, to try cod-liver oil in rickets.

At that date, there was a Dutch family at Tours, under the medical care of the eminent practitioner. One of the children, fifteen months old, became rachitic in an extreme degree. For four or five months, Bretonneau fruitlessly contended with the malady, exhausting the entire series of medications then recommended, when the child's father told him that his eldest son had been cured, in Holland, of the same malady by fish-oil, a popular remedy. Bretonneau advised the same medicine to be given to his young patient: the success was so incredibly rapid, that my illustrious master was quite struck by it.

Encouraged by the result in this first trial, he repeated the experiment on other rachitic subjects; and it was when thus occupied with researches into the action of cod-liver oil, of the good effects of which he had satisfied himself, that he had the pleasure of finding that similar good results had been already obtained by the German physicians whose names I have just mentioned.

Bretonneau informed Dr. Guersant, Professor Jules Cloquet, and me of his curious observations; and then, we, in our turn, administered the remedy to the rachitic children we had to treat. The results were as completely satisfactory as those of which we had been informed. The use of cod-liver oil soon became general; and at the present day, there is not a physician who has not recourse to it under similar circumstances.

How does this medicine act? Is it by specific anti-rachitic virtues such as mercury and iodide of potassium possess in syphilis? I do not believe that it is. Its virtues essentially depend upon fish oil being an analeptic tonic of a superior kind; that is to say, that it acts as a fatty body, and perhaps by being a fatty body combined with different substances which possess the properties of exciting tonics, such as iodine and phosphorus, and combined in the proportions and according to certain modes which chemical analysis might be able to disclose, but which, nevertheless, synthesis cannot reproduce. It resembles, in this respect, all the compound medicaments which we find prepared by the hand of nature: and thus, as I have had occasion to tell you in my lecture on dyspepsia, when speaking of the natural mineral waters, the pretensions of those who wish to substitute pharmaceutical medicines for them is as senseless, in my opinion, as it would be to combine the component parts of wine with a view to produce a substitute for natural wine, even for the worst natural wine. Cod-liver oil, besides being an aliment, is a stimulant perfectly suited to the more or less deteriorated state of the organism.

It does not, however, enjoy a monopoly of these renovating powers. We may perfectly well substitute for it ray oil, herring oil, and the fish oil of commerce. For my part, when I have to treat the children of families whose means require to be considered, I prescribe the oil used by shoemakers, which is much less expensive than either ray or cod oil. However repugnant this oil may appear, the little patients generally accommodate themselves to it as well as to the other oils. I would add in respect of cod oil, that that which is not purified—the brown oil—is far preferable to all the pale kinds, the advertised prospectuses of which proclaim their superiority.

Clinical experience testifies in favour of this opinion; and clinical results are corroborated by those obtained in physiological experiments made in relation to the assimilation of those oils by the economy.

You are aware, that while vegetable oils are either not assimilated, or are only assimilated in so small a proportion that after their reception into the stomach, they become converted into emulsions, and are to a great extent passed by stool, the animal oils are assimilated. There are, however, differences between the two kinds of oil which must be pointed out. The more impure and rancid that the animal oils are, the more perfectly are they assimilated, the more does the digestive canal become accustomed to digest them: on the other hand, the purified oils, such for example, as the pale cod-liver oil, after they have been taken for a certain time, cease to be so well assimilated.

In northern countries, on the shores of the Baltic Sea, where it is a common practice with the people to give to weak children and valetudinarian adults whale oil, and fish oil indifferently, you may be sure that much care is not bestowed on the purification.

Marvellous and unquestionable though the good effects of fish oils are, there are circumstances in which, from the individuals not being able to bear their use, or from their having an invincible repugnance to them, we are obliged to substitute other substances.

There is one substitute, *butter*, within the reach of all, from which excellent results are obtained; but to obtain such results, it is necessary that a large quantity, sixty grammes, at least, be taken in the twenty-four hours.

So as not to shake the confidence of relatives in a medication so simple as butter, I add to it some of the constituents of fish oil. The following is the formula which I generally prescribe:

Take of—

Fine fresh butter,	300 grammes,
Iodide of potassium,	15 centigrammes,
Bromide of potassium,	50 ,,
Chloride of sodium	5 grammes,
Phosphorus,	1 centigramme.

Mix them *s. artem.*

This quantity, spread on slices of bread, ought to be taken in three days.

When children have a disgust for this preparation, fowl fat may be used as a substitute for it, taken in the same quantity, and in the same manner; or recourse may be had to an old practice, still adopted in Scotland and England, of giving fried bacon, or the melted fat of ham spread on bread.

Well smoked ham, eaten raw, is also recommended as a dietetic auxiliary in the treatment of rickets and osteomalacia: its use combined with that of flat beer [*bière non-fermentée*] has been much lauded, particularly in Germany.

The use of fats and animal oils form the basis of the treatment of rickets and osteomalacia. Need I add, that the patients must be placed in the most favorable hygienical conditions?

They must, as much as possible, live in the country, in the open air, and in dry sunny places. Though it be necessary during the period of the increase of the malady to avoid movements, lest fractures be produced, yet at a later period, when the bones have become consolidated, regular exercise is indicated. Saline baths, particularly sea-water baths, will be exceedingly useful.

It is unnecessary to say, that the thing of paramount importance is an aliment essentially tonic and reparatory: but, Gentlemen, in respect of aliment, I must impress on you the necessity of its being varied according to the age of the patients. For very young infants, for those in whom the first dentition is not completed, milk, particularly the milk of a good nurse, must constitute the exclusive food. I attach so much importance to this rule, that I do not hesitate to prolong lactation beyond the usual term. By doing so, and without the use of any other means, I have seen the recovery of rachitic children. When, from the child having been prematurely weaned, it is averse to resume taking the breast, and when the milk of the cow or any other animal causes indigestion, leading to the intractable diarrhœa of which I have spoken to you, I resort to medication with raw meat.

In infants, after the first period of infancy, and in adults, the regimen ought to consist of a varied combination of animal and vegetable food, care being taken that the former predominates, and that farinaceous vegetables are abstained from, as they are of much more difficult digestion than fresh herbaceous vegetables.

LECTURE LXXXVII.

TRUE AND FALSE CHLOROSIS.

False Chlorosis, or Tubercular Anæmia.—Ferruginous Remedies must not be prescribed in False Chlorosis.—Iron arouses the Tuberculous Diathesis, and promotes its manifestations.—The Tuberculous Diathesis ought to be treated by Bitters and Arsenic.—When the Tuberculous Diathesis exists, Fistula in ano and Leucorrhœa ought not to be cured.—False Chlorosis and Syphilitic Anæmia.—The Blowing Sound in Anæmia is Arterial and Simple: in True Chlorosis it is Double, i. e., Arterial and Venous.—Action of the Vaso-motary System on the Production of Vascular Bellows Murmurs.—True Chlorosis is a Neurosis, alteration of the blood being secondary.—Treatment: Hygienical Conditions.—Iron.— CINCHONA.

GENTLEMEN:—You must have been surprised to see me prescribe medications differing so much from one another for several women now in the clinical wards, all of whom may appear to you to be suffering from chlorosis. In point of fact, they are all pale, cachectic, and neuralgic: in nearly all of them, you hear the blowing murmur in the vessels of the neck: and in these seemingly identical cases, I institute very different kinds of treatment.

You perceive, therefore, Gentlemen, that I am far from looking on anæmia and chlorosis as the same disease; and although, as I readily grant, chlorosis is accompanied by profound anæmia, it by no means follows that anæmic subjects are chlorotic. Decoloration of the blood, and modifications in the relations of its elements, are observed in many pathological conditions differing much from one another; and I am convinced that the confusion introduced by some of your teachers into the diagnosis of the diseases which have anæmia as an element in common, daily sacrifices victims.

You see in bed 25 of Saint Bernard's ward, a woman, twenty-two years of age, who pants for breath on making the slightest movement, is gastralgic, dyspeptic, irregular in her menstruation; and in the vessels of whose neck is to be heard the blowing murmur. At the time of her admission to the hospital, it was difficult not to consider her as chlorotic: but she told me that, some time previously, she had had slight attacks of hemoptysis, and that she had been frequently troubled by a short cough, coming on generally after meals. On examining the chest by percussion, with the most scrupulous care, nothing abnormal was revealed. But, by attentive auscultation, it was discovered, that the pulmonary expansion was less in the right infra-spinous fossa than in the left. I heard neither râles nor vocal resonance; and I am convinced, that two months earlier these signs, so equivocal and so little marked, were entirely absent. I was, however, on my guard: and to-day, when you can recognise cavernous râles and gurgling, accompanied by emaciation and disturbance of the digestive and uterine functions, you can make no mistake as to the sad reality.

In bed 3 of the same ward, there is (admitted for the third time), a young girl, with all the apparent signs of chlorosis; and yet, during the eighteen months that she has been in my wards, I have persisted in refusing to give her ferruginous preparations, which some of you wished me to prescribe for her. She also has false chlorosis: at the apex of one lung, she has feeble inspiration; a circumstance which occupies my attention, and alarms me: she has been treated by ferruginous medicines, from which she has always experienced bad effects, and has observed, that the gastralgia and feelings of discomfort have been invariably aggravated by their use.

The influence of preparations of iron on the health of women reputed to be chlorotic is a subject which demands very serious consideration. Speaking generally, it may be said that iron prudently and gradually administered, is well borne by true chlorotic patients. But if in a disease which has every appearance of chlorosis, the physician fail after having varied both the choice and doses of the preparations of iron, he ought to suspect that he has made a mistake; and, if he observe attentively, he will almost always discover that some formidable affection has already revealed its existence by evident signs.

I do not say that iron does not sometimes give an appearance of flourishing health to women threatened with tuberculosis; but in such cases, some signs will present themselves which will show the practitioner that he is on the wrong tack. When in a girl apparently chlorotic, languid, and devoid of energy, the iron rapidly rewakes the muscular power and the appetite, while at the same time it notably accelerates the pulse, and gives rise to a sort of feverish excitement somewhat analogous to intoxication, there arises reason to fear, that a continuance of the iron will induce an outburst of fever accompanied by local disorders, frightfully rapid in their progress.

When a very young physician, I was called to see the wife of an architect suffering from neuralgia, a pale woman, presenting every appearance of chlorosis: I prescribed large doses of preparations of iron, according to Hutchinson's method of treating neuralgia. In less than a fortnight, there was a complete change: the young woman acquired a ravenous appetite, and an unwonted vivacity: but her gratitude and my delight did not last long. The excitement soon became fever: and the restored color of the cheek became every evening after dinner more ardent than it had been when she was in good health. A short cough supervened; and in less than a month from the commencement of the treatment, there appeared signs of phthisis which nothing could impede.

The first case of galloping consumption which I had to deplore in my practice, occurred under nearly similar circumstances: the patient was a girl of fifteen, who after a mild attack of dothinenteria fell into a state of anæmia and prostration, which I considered chlorosis. I administered ferruginous remedies, which rapidly restored her to florid health: and although there was nothing in the family history to lead me to fear the coming calamity, she was simultaneously seized with hemoptysis and menorrhagia, and died two months afterwards with the symptoms of phthisis, which had advanced with giant strides.

I do not blame the iron for having caused this calamity; but I do blame myself for having cured the anæmia, a condition, perhaps, favorable to the maintenance of the tuberculous affection in a latent state.

Nevertheless, Gentlemen, inasmuch as under the influence of bitters, arsenic, hydrotherapy, and sea-water baths, I obtain equally favorable results in women who seemed to me to have been in conditions quite the same as those which I have just been describing; and, inasmuch, as I have not seen produced in them that general excitement, the prelude, cause or effect, of the tuberculous dissolution, I am constrained to impute to iron some of the evil consequences which I had to deplore.

For many years past, I have looked on it as a duty, when I have had to do with persons hereditarily predisposed to tuberculosis, not to push too far a medication under which all the functions seemed to regain a powerful energy; and though in these same persons, physicians less timid than I am, dare to seek and to obtain results which I dread, I avoid their practice, because under it I have often seen diseases show themselves which might otherwise have remained for a long time in the germ.

Gentlemen, a book has been written on the advantages of bad health; and without guaranteeing all that may have been said on that subject, I would wish to appropriate some of it, and tell you that you will avoid distressing occurrences in your practice by allowing patients predisposed to tuberculous disease to retain maladies from which it may seem easy and opportune to deliver them. It has been impressed upon you by your surgical professors that it is dangerous to cure anal fistula in tuberculous subjects: and it is now a long time since I laid down for myself the rule, not to interfere in young women predisposed to tubercle with leucorrhœa, so common in them, sometimes so inconvenient, and so easily cured. In cases in which I have refused my consent to such patients being treated for this indisposition, and in which other practitioners, more venturesome, have acted otherwise, there have soon supervened accidents till then retarded.

In respect of fistula and leucorrhœa, I have no objection to the theory of revulsion being invoked, yet it has seemed to me that the relative debility of the patients was to them a protection against the sudden outbreak of tuberculous accidents. The older I grow in the practice of my art, the more convinced do I become, that in the same family in which the hereditary taint of scrofula exists, the women who are anæmic, or affected by indispositions which maintain them in a state of precarious health, pay their debt to hereditary taint at a later period than those who are seemingly in the enjoyment of the most complete health.

Gentlemen, I am well aware that very few practitioners concur in these views; and still better do I know the extent to which I have been subjected to the criticisms of those who make use of iron so frequently (and as I think) so inopportunely; but my conviction, far from being lessened, is confirmed more and more every day.

Let us now, Gentlemen, recall the case of a woman, aged thirty-two, who lay in bed 22 of Saint Bernard's ward. On admission, she was in a state of extreme anæmia, having blowing sounds in the vessels, chronic diarrhœa, and an exceedingly profuse leucorrhœa. I fought long, but fruitlessly, against the abdominal affection. One day, I moderated it—the next, it reappeared. For four months, I used, with great perseverance, all the therapeutic measures with which I am in the habit of successfully treating diarrhœa. Nevertheless, a temporo-facial neuralgia supervened, an event not at all surprising in a person so exceedingly anæmic; but the neuralgia had this peculiarity, that it returned every evening, increased in severity during the early part of the night, and ceased at day-break. This nocturnal return of the malady made me suspicious, and led me to fear the existence of constitutional syphilis, with which, notwithstanding her protestations to the contrary, I felt convinced she was affected.

Some time afterwards, a very painful exostosis on the crest of the tibia declared itself. I no longer regarded the denials of the patient. I prescribed Van Swieten's liquor (bichloride of mercury); and you have seen with what rapidity the health of this woman, so seriously compromised, was reestablished. You have seen her color return under the influence of mercury, a medicine which so radically alters the crasis of the blood in healthy subjects.

Nearly at the same time that this woman was under treatment, you saw, in bed 16 of our nursery ward, a young woman who was pale, and had all the symptoms of chlorosis. She had no sign of venereal disease: but the child she was nursing had syphilitic symptoms, and hypertrophy of the liver: it was even paler than its mother. She had been uselessly treated by ferruginous preparations. Mercurials, followed by iodide of potassium, restored her to the appearance of perfect health. Bear in mind, in relation to this subject, the interesting researches of Dr. Grassi regarding the blood of persons affected by syphilis. This distinguished chemist has demonstrated, by numerous analyses, that in the secondary period of syphilis, there is a diminution in the number of the blood-globules.

I have been anxious to place before you these facts, to let you see that a multiplicity of causes may alter the constitution of the blood in such a manner as to simulate chlorosis; and I have done so for the special purpose of guarding you against the hackneyed treatment by ferruginous medicines, which, in the majority of cases, is insufficient, is, moreover, sometimes useless, and more frequently dangerous.

When my honourable colleague, Dr. Bouillaud, first called the attention of practitioners to the murmur which he called the "*bruit de diable*," the "*bruit de souffle musical, et à double courant*,"[1] he did not suspect that it would become with many practitioners a frequent cause of perilous diagnosis.

Look, Gentlemen, at the patient who now occupies bed 29 of Saint Bernard's ward, whose malady is true chlorosis. Study in this case, the vascular murmurs, and specially study the other phenomena which constitute this curious disease. Let me, in the first place, speak of what takes place in respect of the vessels.

On applying the stethoscope below the middle of the clavicle, we have a rather dry blowing murmur, accompanying the first sound of the heart. But during the ventricular diastole, the murmur assumes a more musical character, louder, and resembling the purring of a cat when it is being caressed, or the noise of a spinning-wheel. Between the first and second sounds of the heart, the murmur never altogether ceases. The name given by Dr. Bouillaud, viz. "sustained blowing-sound" [*bruit de souffle avec renforcement*] is, therefore, appropriate; but it is important to remark, that the continuance of the sound takes place during the cardiac diastole. Along with many other physicians, I believe that the first sound is in the arteries, and the second in the veins. On compressing, by the application of a thread, the lateral part of the neck, above the point where the stethoscope is applied, in such a way as to interrupt the current of venous blood, we find that the second sound ceases.

Whatever there may be in this explanation, it appears to me that there are two classes of blowing sounds of the neck; viz. the simple sounds, purely arterial, and the double-current sounds [*bruits à double courant*] so well investigated by Dr. Bouillaud. The first belong to anæmia, whatever may be the cause of the anæmia: the others are peculiar to chlorosis. They are so decidedly chlorotic sounds, that they precede or follow the most ordinary manifestations of chlorosis. We have at this very moment, in bed 3 *bis* of Saint Bernard's ward, a young woman who is typically chlorotic. You recollect that when she was admitted to the hospital, she presented, with the utmost exactitude, all the symptoms of chlorosis. Ferruginous treatment rapidly reestablished her health: the complexion and the mucous membranes regained color: the veins were traced out by blue lines: and all the functions were restored in their integrity. However—as I have often asked you to verify by your own personal observation—the double-current blowing sound still continues in the vessels of the neck, and though it is not so strong as it was two months ago, it is not the less distinctive: on the other hand, in all the anæmic women in the wards, you can only hear the simple murmur accompanying the first sound.

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[1] Bouillaud: Traité Clinique des Maladies du Cœur. T. I, Paris; 1841.

It therefore follows that, in chlorotic subjects, the vaso-motor nervous system is modified in a manner altogether special, and that this modification is, to a certain extent, independent of the constitution of the blood. It bears no relation to the greater or less number of the blood-globules, because, in the first place, anæmic subjects seldom have the double-current blowing murmur, and, secondly, because chlorotic subjects, after the constitution of their blood is restored to its normal state, long continue to present this sign.

When, as the result of clinical observation, I was led to think that differences existed between the anæmic and chlorotic blowing sounds; and that these sounds may continue for a variable period after the cure, I was not acquainted with the conclusions arrived at by Dr. Peter, as stated in an unpublished memoir on this subject.

In this work, Dr. Peter has established, upon a basis of sixty-three cases, that the vascular blowing sounds are not entitled to retain that importance which has been assigned to them in the diagnosis of anæmia and chlorosis. It has indeed been shown by the chemical analyses of MM. Andral and Becquerel, that vascular blowing may be perceived in cases in which the number of blood-globules is not below the numerical physiological average. As, on the other hand, it has been shown by Dr. Peter, that the symptoms of anæmia and chlorosis may exist without there being any vascular blowing, is it not natural to conclude, that its presence or absence is only of secondary importance in the pathological conditions which we are now considering?

Dr. Peter has also shown, that the blowing sounds may disappear and reappear within a few hours, evidently, therefore, without the crasis of the blood having undergone any sensible modification. Since then, vascular blowing may be heard when there is a normal constitution of the blood—when there exists the functional disturbance of anæmia or chlorosis—since vascular blowing may appear and disappear during the same examination without there being any possible alteration in the condition of the blood, we must attribute this sound, in a certain number of cases, to something else than the state of the blood. May not the sound be caused by a peculiar state of the vessels, by contraction of their parietes? This is a view which brings us back to the opinion of Lænnec, who attributed the vascular blowing heard in the hypochondriac regions to spasm of their vessels, a spasm which he thought, and which I think, depends upon the action of the vaso-motor system of nerves.

Dr. Peter, with Lænnec, believes in vascular spasm: with M. Chauveau, he believes that the phenomenon of the contracted column of blood [*la phénomène de la veine fluide*] is the condition which occasions vascular blowing; or in other words, that the spasm of the vessel produces constriction, which constriction in its turn produces the contracted column. As you know, according to the experiments of Savart, there is always produced a contracted column when a liquid in traversing a tube passes from a constricted to a wider portion.

From the reasoning and observations of Dr. Peter, it follows, that the vascular blowing sounds do not possess the semeiotic value hitherto attributed to them: they are by no means pathognomonic of anæmia, and possess a value which is only relative. For, adds Dr. Peter, as the wall of the vessel derives its nervous supply from the vaso-motor system, nervous people are subject to vascular spasms, and as anæmic people are in a high degree nervous, it follows that it is principally among them that vascular blowing sounds are met with—and here vascular blowing is a very indirect proof of anæmia. But as a person may be anæmic without having these vascular spasms, and have vascular spasms without being anæmic, it equally follows, that vascular blowing cannot be a direct proof of the existence of anæmia.[1]

In an interesting discussion, raised before the *Société Médicale des Hôpitaux* by Dr. Parrot, that able clinician also reduced the usually accredited value of vascular blowing as a sign of an altered state of the blood. He also believes, that it is the wall of the vessel, and not the condition of the circulating fluid, which produces the blowing sound. He also observed that these sounds are fugitive, appearing and disappearing during even the same examination, according to the position of the patient: he adds, that they are usual in children, common in aged persons, and almost invariably met with in the nurses at the *grand bureau,* who are generally robust women, recently arrived from the country, and free from all signs of debility or nervousness.

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[1] PETER:—*Gazette des Hôpitaux*: 1867. This theory of Dr. Peter was formally stated in 1863, in the second edition of these Clinical Lectures.

In explaining the production of the vascular blowing sounds, Dr. Parrot attaches very little importance to the condition of the blood: he believes that the sounds are produced in the veins and not in the arteries, and that they are due to insufficiency of the venous valves, an insufficiency which does not admit of any doubt in respect of the valves of the internal jugular vein. The greater frequency of vascular blowing sounds on the right side is attributed by Dr. Parrot to the almost rectangular passage of the vessels on that side, and to the shortness of the passage to the heart.[1]

Dr. Parrot does not go so far as to allege that vascular blowing is valueless in the diagnosis of anæmia and chlorosis: but he says, that to give it a decided value, it must be very intense and accompanied by a purring sound.

In his beautiful work on anæmia, Professor G. Sée, who locates vascular blowing in the arteries, also attributes it to the nervous influence in operation from the time that the blood has begun to be altered. There is diminution of the tension of the blood consequent upon relaxation of the vessels. The blood flowing more irregularly than by the capillary extremities, the pulse becomes quicker, and a maximum abnormal sound is produced. It is by the fluid column that Dr. Sée, in like manner, explains vascular blowing. He is also of opinion that the material conditions which produce vascular blowing sounds are permanent weakness of the heart's action, feeble tension of the arteries, the brisk and accelerated flow of blood which passes with ease from the arterial trunks into the minute arterial branches. In the fever of the pyrexiæ, there is a constant fatigue of the muscles of the vessels, and their tension is always enfeebled; and then, we hear vascular murmurs: they are never, however, so distinct as in chlorosis, because the blood is not impoverished in globules. Blowing sounds are likewise produced in hysteria and hypochondria: in these affections, they are derived from a direct excitation of the heart, but which is not sustained by any alteration of the blood.

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[1] PARROT:—*Archives Générales de Médecine:* T. ix, Juillet, 1867.

Arterial blowing, then, does not possess any absolute diagnostic value, says Dr. Sée, and is in itself insufficient to establish the existence of chlorosis, particularly when the disease does not manifest itself by other signs.[1]

Dr. Potain[2] also is of opinion, that vascular blowing is not a sign, certain and pathognomonic, of anæmia; but he believes that the composition of the blood may have some influence on its production, and that, consequently, it is not without some semeiotic value. By experiments, equally ingenious and delicate, Dr. Potain has shown, that three vascular phenomena are observable in the region of the heart, viz. a movement, a thrill, and a sound. The movement consists of two slight upheavings followed by two more considerable sinkings, succeeded by a slow ascent of the region. These movements are due to the veins; and the application of the sphygmograph shows that the first upheaving immediately precedes the contraction of the ventricle, and corresponds to the systole of the auricle: the second upheaving is due to the systole of the ventricle. Thus, the two successive elevations of the vessels are due to successive contractions of the right auricle and right ventricle. The collapse of the veins corresponds with the diastoles: the first collapse is due to the diastole of the auricle, and the second, to the diastole of the ventricle. Though the thrilling is intermittent, it is produced during the vascular collapse, that is to say, during the cardiac diastole, a fact which harmonises badly with the theory of Dr. Parrot, who attributes the thrilling, as well as the blowing, to insufficiency of the valves of the veins; for in that case, the thrilling ought to take place at the time of the upheaving of the vessel and to be coincident with the cardiac systole. Dr. Potain, in relation to the vascular murmurs, without rejecting Dr. Peter's spasm-theory, relates experiments which he performed, and from which it appears, that the greater the fluidity of the circulating liquid, the more intense is the blowing which it produces in the tubes wherein it circulates: thus, the blowing sounds are very loud when it is water or serum which circulates, and they cease altogether when these fluids are replaced by blood. But the greater the fluidity of the liquid, the more rapid is its circulation; so that the whole discussion resolves itself into a question of rapidity of circulation. As the blood is less dense, and more fluid, when there is a diminution in the number of red globules, it follows, that the rapidity of the passage of the liquid must be augmented, and the conditions be realised which produce vascular blowing. This is what occurs in chlorosis: it is, therefore, impossible to deny, that the quality of the blood possesses an influence upon the production of the blowing sounds.

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[1] Sée :—Du Sang et des Anémies: *p. 202 et passim:* Paris: 1866.

[2] Potain :—*Gazette des Hôpitaux.*

The exposition which I have now made shows, that we must not, as hitherto, exclusively attribute vascular blowing to the state of the blood. The causes of the phenomenon are much more complex. Fluid blood flows more quickly, and fluidity is a condition favorable to the production of vascular blowing; but fluid blood is less rich in globules, and being less rich in globules, it is less nutritive and less an excitant of the nervous system. It is at this point that the too much neglected action of the walls of the vessel plays its part. It matters little whether the parieties of the vessel become paralytically relaxed, or spasmodically contracted: the important point is the occurrence of nervous disturbance—of a temporary and fugitive character, like everything which pertains to life—a disturbance which temporarily modifies the circulation in such a way as to produce a contracted column, and, as a consequence of that, a blowing vascular sound. We can thus understand how it is, that the blowing sound may appear and disappear in the course of the same exploration, as observed by Drs. Peter and Parrot. Moreover, the strength of the blowing sound is greater, if, in addition to the nervous state of the vessel, there exist greater fluidity of the blood.[1]

In his conclusions, which are perhaps too absolute, Dr. Peter has been specially anxious to place in relief the action of the living solid, the wall of the vessel, the contractile wall, supplied by nerves from the great sympathetic, and to oppose its action, modifiable to an infinite degree within a space of time relatively very short, to the action of the blood, which cannot change sufficiently quickly to explain by itself the rapidly changing blowing sounds in the vessels. I am ready to admit with Dr. Peter, that in some nervous persons vascular blowing exists irrespective of an alteration of the blood; but that which is only observed in them accidentally is the rule in the hydremic and chlorotic.

Bear in mind that, in exophthalmic goitre, continuous re-enforced vascular blowing is heard, particularly during the paroxysms of that disease. These paroxysms appear suddenly—most frequently, consequent upon strong mental emotion, and although the blood cannot, within a few minutes, undergo extensive changes in its constituent parts, it is during the paroxysms of Graves' disease, as well as in acute or chronic chlorosis, that vascular blowing is the result of a modification of the contractility of the vascular system.

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[1] For information in relation to this interesting discussion, *see* the *Bulletin de la Société Médicale des Hôpitaux:* also, the excellent article, CHLOROSE, by DR. LORAIN in the Dictionnaire de Médecine et de Chirurgie Pratiques; T. VII, Paris: 1867.

I think, however, that chlorotic blowing ought always to be carefully sought for; and the reason for my laying before you the minutiæ of the auscultation of the vessels of the neck is because they are of very great importance. While in the majority of cases of anæmia, ferruginous remedies are not generally trustworthy, and are sometimes dangerous, they possess an almost invariably useful and rapid influence upon chlorosis.

From what I have now said, Gentlemen, you have no doubt perceived that I am disposed to class chlorosis with nervous diseases.

Let us, for a moment, leave out of consideration the constitution of the blood, and inquire by what other phenomena than paleness of the tissues, the disease shows itself. These phenomena have an almost exclusive bearing upon the nervous system. The intelligence, the senses, the muscular movements of animal and organic life, are very much modified. Chlorotic girls generally experience perversions of the understanding, and I am acquainted with many such cases. They become irrascible, and odd in their behaviour; the mental disturbance sometimes goes so far as to be really insanity. If we examine very carefully into the state of the skin, we find that its sensibility is deficient in many places, while in others, it is occasionally morbidly increased. I never examine a chlorotic woman in your presence without interrogating her as to neuralgic pains; and you may have perceived, how unusual it is to find a chlorotic patient who does not suffer from them more or less. In these patients, facial is of all forms of neuralgia the most common: very often it alternates with intercostal neuralgia, and with neuralgia of the stomach, liver, intestines, and uterus.

Spasmodic affections of the locomotor system of animal life are very frequent; and you know how often hysterical convulsions are met with in chlorotic women. Palpitations of the heart, and spasms of the stomach intestines and uterus occur in nearly all chlorotic subjects.

The disturbance of the nervous functions produces great modifications of the secretions. The secretions of the stomach are altered in their chemical composition; hence we have pyrosis, pica, &c.: the secretions of the liver and kidneys are sometimes suppressed, and sometimes augmented, giving sufficient evidence of the existence of that nervous disturbance of which I have been speaking; and the great ovarian secretion, which constitutes one of the most important secretions of women, is very often suppressed along with menstruation, of which it is the consequence.

But, Gentlemen, amenorrhœa is not always the accompaniment of chlorosis. Many years ago, I published a work upon *ménorrhagic chlorosis*, in which I showed that in virtue of exceptional tendencies, very difficult for me to appreciate, the menstrual flux becomes exceedingly profuse, and augments as the malady progresses. In such cases, ferruginous medication is quite as powerfully remedial as in normal chlorosis or amenorrhœa.

Gentlemen, we are in the habit of regarding chlorosis as a malady which is not serious; and in so doing, it appears to me, we take too little into account a general condition in which we see supervening great disturbance of the economy. I have long looked upon chlorosis as a serious affection. It has the peculiarity of leaving an almost indelible impression; so that when a young woman has been very chlorotic, she remembers it for nearly her whole life. If you interrogate women who have had several attacks of chlorosis, and have reached the catamenial decline, you will find that they present neuropathic phenomena which almost never leave them, however variable may be the forms which they assume. And this is observed when the blood-lesion has long previously been repaired: sometimes even, plethora may be observed. This is a farther proof, that chlorosis must be regarded as a nervous disease caused by alteration of the blood, rather than as a cachexia causing nervous disorders.

I need not now recall to your recollection experiments recently instituted by the ablest physiologists demonstrating the influence which the different functional disturbances of the nervous system stamp upon the secretions, as well as upon the composition of the blood. It is easy to understand, that when the essential functions of an organ of hematosis, such as the lungs liver or spleen, are altered, the composition of the blood must undergo great modifications.

This influence of the nervous system sometimes makes itself felt with wonderful rapidity. Recall to your recollection, a young woman, bed 32, Saint-Bernard's ward, a patient who has already come twice into our wards to be treated for Saint-Vitus's dance. When in the enjoyment of very good health, she received a sudden fright during the night. Next morning, her health was affected; and four days later, she came into our wards with all the signs of confirmed chlorosis. In bed 3 *bis* of the same ward, we had a girl of eighteen, who, also, consequent upon, and some days after, a violent mental emotion, became chlorotic. These cases show you how little importance can be attached to the primary state of the blood; and how important it is to place anæmia only among the secondary causes of chlorosis.

Amenorrhœa, so common in chlorosis, sometimes announces the beginning of the disease. In other cases, a perfectly healthy young woman becomes suddenly chlorotic, when the catamenia, which had commenced, are suppressed, in consequence of a chill or some great mental shock. Certainly, in such a case, the dyscrasia of the blood cannot be considered as the result of a hemorrhage; and as it also occurs in certain cases of anæmia, it can be imputed only to nervous perturbation. On the other hand, we see young girls remain chlorotic for a long period although the most rational treatment has been employed, and cease to be chlorotic on the appearance of the menstrual hemorrhage, that is, when the anæmia ought to be increased by the loss of blood. The explanation of the case is this: —on the cessation of the general nervous disturbance, the normal secretions are reestablished, because the cause of the chlorosis itself has ceased.

From the remarks which I have made, you may pretty well conclude that chlorosis is not always so easily cured a disease as has been supposed. In the clinical wards, you have seen some young women cured very quickly, whilst in others, we have had to wait a long time ere we saw the results of the treatment. In fact, to conduct this malady to a happy issue, a combination of favorable conditions is required, not always easy to attain or meet with in hospitals, nor even in private practice.

I have told you, Gentlemen, the part which the nervous system performs in chlorosis: you can understand, therefore, how important it is to withdraw the patient as much as possible from all deleterious mental influences. Though I have endeavoured to prove that anæmia is an effect and not an essential element of chlorosis, I am not the less of opinion that the dyscrasia of the blood, under whatever circumstances produced, has a tendency to induce chlorosis, and that it would be impossible, whatever medication were employed, to cure a patient unfavorably placed in respect of regimen and residence. It is vain to administer iron and bitters, if a subject exposed to paludal miasmata, lose daily, under the influence of that morbific cause, all that is gained by the ferruginous treatment. It would be the same if (as often happens) the patients refuse to take exercise in the open air, and obstinately remain shut up in obscure apartments where they become etiolated. It would also be the same, were profuse menstruation, or frequent attacks of epistaxis, to destroy as much as the remedies restore. Finally, if the supply of food be insufficient, the alteration of the blood will increase; and thus, as you perceive, we shall always be turning round in the same vicious circle.

But it is easy to foresee the difficulties presented by the *circumstances apparently accessory.*

Though we can remove patients from countries where intermittent fever prevails, it is not so easy to contend against paludal infection when once established in the system. It then becomes necessary to treat the intermittent fever with the same energy and perseverance demanded in paludal maladies which have long dominated in the system. And it is only then, when we have mastered the intercurrent disease, that we can beneficially institute our treatment for the chlorosis.

As to the etiolation, the result of deprivation of light, to which, from perverted inclination, the young patients often condemn themselves, it is necessary to arrest it with the least possible delay, and to employ severe measures to accomplish this object should the weakness of relations render them necessary by placing obstacles in our way.

Uterine hemorrhages and epistaxis, as I have told you, are sometimes the effects, rather than the causes, of chlorosis. Although, in general, ferruginous medication may of itself be sufficient treatment, it too often happens that we gain nothing by it, in consequence of the constantly renewed hemorrhages not being compensated for by the reparation derived from the iron. These are the cases in which the powder of cinchona, extolled by Bretonneau, renders services not to be expected from any other remedy; and although I do not know to what principles cinchona owes its potent properties, I can testify not the less confidently, that in menorrhagia and obstinate epistaxis, powder of cinchona, taken internally in doses of from two to four grammes, daily, or two or three times a week, quickly moderates and rapidly cures the hemorrhage.

This therapeutic agent is much superior to rhatany, tannin, and the mineral acids, as I have had frequent opportunities of seeing in my clinical wards.

But if, as sometimes happens, medicines do no good, we must not hesitate to employ surgical measures, however disagreeable that course may be. Plugging the nasal fossæ by the different means with which you are all acquainted will arrest the bleeding, should you have failed to attain that object by the use of astringent injections; but you can understand that injections are useless in menorrhagia, as the medicinal agent does not come in contact with, and indeed cannot reach the mucous surface of the uterus. It is then that plugging becomes indispensable: and however disagreeable it may be to have recourse to such a proceeding in a young girl, and however painful it may be, we must not hesitate to employ it, because it is the only treatment which has any chance of being successful.

I had to treat a young lady of nineteen who was chlorotic to an extreme degree, and likewise subject to menorrhagic attacks which were at first moderate, but ultimately became intractable. The loss of blood, sometimes so great as literally to traverse the mattress and fall on the floor, obliged me to use plugging: it succeeded wonderfully; and I was able by ferruginous medicines to restore this patient to perfect health. She quitted Paris; and had subsequently a recurrence of the symptoms. Upon this occasion, her family shrunk from consenting to a means of treatment to which the young lady had an intense repugnance, and to the carrying out of which a difficulty was perhaps presented by the youth of the attending physician. Death from hemorrhage supervened, as internal remedies failed to arrest the hemorrhage.

My friend Dr. Campbell has often mentioned to me the wonderful successes which he has obtained in formidable hemorrhage supervening after delivery, by administering enormous quantities of spiritous liquors according to the practice of many English practitioners. He has ordered the administration, in table-spoonfuls, of as much as a litre of brandy or rum in the twenty-four hours. He has given, at the same time, sherry, Madeira, and Malaga wines. Strange to say, the women bore the large doses of alcohol without experiencing the slightest inconvenience, so long as they were under the influence of these hemorrhages, whilst as soon as they recovered they were unable to take the smallest quantity without becoming drunk. Professor Dubois and I have seen similar cases.

May not this treatment, however extraordinary it may seem, be as much indicated in the menorrhagia of which I have just now been speaking, as in profuse hemorrhages consequent on delivery.

A few minutes ago, I spoke to you on the subject of regimen: I remarked, how important it was that the patient should have suitable alimentation: but here, an insuperable difficulty often arises. The disease not only engenders dyspepsia, but also a capricious appetite, a disgust for the most substantial fare, and an insensate craving for things generally regarded as very bad. There are some chlorotic girls who would rather die from hunger than eat the usual food of other people.

In such cases, Gentlemen, we must not hesitate to submit to one of those therapeutic capitulations so often demanded in the exercise of our art. I generally pay very little attention to the nature of the food, provided it be digested. I sanction without scruple the use of articles reputed to be very indigestible, such as radishes, salad, hardly ripe fruit, strong tasting cheese, very sour wine, vegetables, highly spiced pork-meats, acid beverages, and spirituous liquors. But if I am thus indulgent, I expect a concession in return which is, that this strange aliment be varied. In relation to this point, Gentlemen, allow me to make a short digression, not altogether irrelevant.

Man and the lower animals are so constituted that they allow themselves to follow the same routine in diet as in other matters; yet there are many things in which a change, even when it is a change for something worse, is accepted by the economy, not only without injury, but sometimes with benefit. The great effect produced by a mere change of residence is astonishing: the person who daily takes suitable exercise, but whose regimen is sometimes not very good, and who breathes a less pure air and takes the same exercise, experiences a sort of transformation and feeling of betterness, which are simply the results of a new excitement in the economy produced by impressions to which it has not been accustomed.

The same observation is true in respect of regimen. We know that the stomach is easily tired by a constant repetition of the same articles of food, and that its functions are favorably influenced by change of regimen. On the other hand, though experiment on domestic animals which it is wished to fatten, proves that the same amount of equivalents of nutriment produces proportionately better results, the more the elements are diversified, yet experiments instituted in the human subject demonstrates that, though in our ordinary meals, we become satiated by a determinate quantity of food, which cannot be exceeded without producing some disorder in the digestive functions, we may, at a banquet of numerous and of varied dishes, take with impunity a double quantity of aliment.

Gentlemen, pardon this digression which I believed necessary to enable you to understand fully, that though we may condescend to allow chlorotic women to eat articles reputed indigestible, all objection ceases provided we arrange so that in every meal there is a multiplicity and variety of dishes. It is in this way that we can arouse the digestive powers, and impart to the blood, with even an insufficient alimentation, some of the constituents which are wanting, and prepare the way for therapeutic agents of which I shall forthwith have to speak to you.

The concessions of which I have been speaking must not be regarded as mere acts of complaisance. When we see chlorotic girls easily digesting aliments which they would have rejected some years previously, the question arises, whether, under the influence of the great disturbance of the nervous system which accompanies chlorosis, new aptitudes do not replace those which are lost, in such manner, that the digestive organs find themselves accidentally in functional harmony with the aliments better suited to the organs of other animals.

Marriage is one of the hygienical measures recommended to chlorotic girls, regarding which physicians are often asked to give an opinion. The strange idea, after germinating in the heads of some physicians, has become popularised, that the erotic instincts are more developed in chlorotic than in other women.

I willingly admit that puberty indicates a woman's fitness to conceive; but I deny that this fitness arouses in her instincts similar to those which it arouses in a man. In our order of society, young girls are as remarkable for chastity in thoughts as in actions; and when chlorosis declares itself in married women whose confidences we obtain, we often learn that their sexual appetites have diminished in proportion as the disease has progressed. That, certainly, does not absolutely prove that marriage would be useless, but it at least seems to indicate that the acts which are the usual consequences of matrimony are little necessary, and may be instinctively repugnant to chlorotic women.

I grant that it is not convenient to cause a young woman to defer too long the duties of maternity, for which she was created; but that is a very different thing from adopting the common notion, that marriage is indicated as a means of cure in a multitude of maladies. Suppose that a girl who from childhood has had horrible scrofulous or dartrous sores, is seized with epilepsy, hysteria, or intermittent mania—is not that the doleful dowry which she would bring to a young husband, but which would be carefully excluded from the stipulations of the marriage contract?

I now come to speak of the treatment by the administration of pharmaceutical substances.

Iron occupies nearly as important a place in the treatment of chlorosis as cinchona in the treatment of intermittent fever; but there is great need of young physicians forming correct views as to the doses and modes of administering ferruginous medicines. We know very well that chlorosis is a disease which has a great tendency to recur: and, as I have told you, a woman who has been seriously chlorotic for a long period retains traces of the formidable neurosis as long as she lives. It is, therefore, an essentially chronic disease; and *chronic diseases must have chronic therapeutics.* That is one of the most elementary precepts in medicine; so that the administration of iron ought to be persevered in for a long period, and frequently resumed. The length of the intervals during which the use of the iron is discontinued will be the more prolonged the more perfect the state of health. After achieving a victory, we must not go to sleep. When, after six weeks or two months of treatment, the colour and the menstrual functions are restored, we must proceed further than the apparent cure, and recommence after intervals of two or three months, and continue to do so during three consecutive years, should the chlorosis have been of old standing, and have left a deep impression on the economy.

It is rather difficult to state with precision the doses of iron which are necessary for the cure of the malady; and in respect of dose, perhaps greater diversities occur than are observed in other diseases. We all know that in treating tertiary syphilitic affections, we see one individual get rid of violent osteocopic pains in a few days by taking very small doses of the iodide of potassium, while another, who seemed to be precisely similarly affected, will not obtain relief till he has taken doses of the iodide ten times as strong, and during a much longer period than in the other case. The remarks now made are applicable to a multitude of medicines besides iron. Iron is, however, one of those medicines by which similar results are obtained by the greatest diversity of dose. Some chalybeate waters which, like those of Pougues, Spa, and Schwalbach, scarcely contain a few centigrammes of the salts of iron to the litre, sometimes cure chlorosis more quickly than steel filings, black deutoxide of iron [*l'ethiops martial*], or subcarbonate of iron [*les safrans de Mars*], which are given in doses of some grammes daily. Let me state, however, that, in general, ferruginous preparations ought to be administered at each meal in doses of from fifty centigrammes to one gramme; and that the only limit to the dose is the tolerance of the stomach.

Pharmacy has in preparations of iron a luxurious abundance, unfortunately, to some extent, created by commercial speculation. Every inventor of a new salt finds good reasons for celebrating its superiority. But, after long trial of the old and new preparations, both in my private practice and under your observation in the clinical wards, I feel convinced that no preparations are so well suited for administration in the form of pill as the filings and the subcarbonate, which may be made into a mass with the extracts of cinchona, chicory, wormwood, or sometimes with rhubarb, provided certain special indications are attended to. Extract of wormwood is a useful excipient in the case of women who have amenorrhœa combined with dyspepsia; and the majority of cases are of this nature. When there is obstinate constipation, the addition of a small quantity of the extract of rhubarb, the exact dose of which cannot be specified, may be of great service. With women in whom there are none of the accidents inherent to chlorosis except want of appetite, the soft extract of cinchona generally agrees well.

When I do not give ferruginous preparations in the form of powder or pill, the liquid form which I prefer is the syrup of the ammonio-citrate of iron in the form of fifteen grammes of the salt to five hundred of syrup. From one to four teaspoonfuls may be taken at each meal.

Gentlemen, you have seen me several times prescribe ferruginous preparations to chlorotic patients who were unable to bear them. In general, iron is borne well, at the first start, in chlorosis; and as I remarked at the beginning of this lecture, it is badly tolerated in many cases of anæmia: and we must be on our guard when the economy does not support it. But although the diagnosis has been carefully established, and the indication is exact and precise, iron may be badly supported; and then it is necessary to use artifice to make a useful remedy be tolerated. Iron, as I have already said, like most medicines, is most suitably administered during a meal. Even, however, when this precaution is taken, the stomach seems sometimes to refuse it, gastralgia increases, dyspepsia becomes more serious: diarrhœa occurs sometimes; and, still more frequently, there is invincible constipation. Then it is, when diarrhœa and gastralgia dominate, that minute doses of extract of opium may be very advantageously combined with the iron: then too, belladonna in very small doses cures the gastralgia, and, with still more certainty, removes the constipation.

The administration of iron ought from time to time to be interrupted, and cinchona wine given in place of it. Sea-water baths, hydrotherapeia, and sulphurous baths are very useful adjuvants.

It often happens, Gentlemen, that recovery from chlorosis takes place without any medicinal intervention: and sometimes you have seen me seek to cure a case of this malady by the peroxide of manganese, when I have failed with ferruginous preparations. Under the influence of these different medications, restoration of the normal constitution of the blood takes place, and disappearance of all the symptoms peculiar to pale-faced chlorotic patients.

The remarks which I have just made must suggest to your minds some doubt as to the part which iron plays in the treatment of chlorosis. You ask yourselves whether it really is from the ferruginous remedy that the blood derives the very small quantity of iron which it requires. Animals in a state of health reconstitute all the elements of the blood from their food; and when we consider how small is the quantity of iron contained in the entire mass of blood in the body, we can easily see that there will always be enough of it in the ingesta.

When a fecundated hen's egg receives the influence of heat in incubation, each of the most intimate elements which enter into the composition of the white and the yolk is distributed to the different parts of the animal, in virtue of vital affinities inherent in the organic molecules. The calcareous salts form the bones and the feathers. The albumen, modified in its composition, constitutes the muscles and the blood: the small quantity of iron found in the newly laid egg concretes to form hepatic globules simply in virtue of nutritive activity: that this take place, it is sufficient that the egg be endowed with life, be sound, and be placed in certain conditions.

We can understand, that in the same way, the living organism, in a healthy woman, can derive from the different alimentary substances, the majority of which contain iron, the elements requisite for the constitution of the blood and muscles; and, as I have just been saying, the quantity of iron contained in the economy is so small, that the usual aliment is amply sufficient for the necessary reparation. It is, therefore, not indispensable that the ferruginous therapeutic agent should furnish to the blood its deficit of iron. It is enough that the ferruginous remedy put the organs into that condition of health which imparts the power necessary for the assimilation of the iron in the food, a power similar to that by the exercise of which the organic elements of the chicken assimilate the iron contained in the egg.

You perceive, Gentlemen, the point to which we are led by the theory of those who desire to find the principal constituents of the blood in the medicine. In syphilitic anæmia, and in paludal anæmia, we must accord respectively to mercury and cinchona, a part similar to that which iron plays in chlorotic anæmia. Now, I have shown you, and, moreover, you know very well, how powerfully mercury and cinchona act in reconstituting the blood, and, consequently, in restoring to it the iron in which it is deficient. Therefore we may, indeed we ought, to consider iron as the specific for chlorosis, just as mercury and cinchona are respectively the specifics for syphilis and marsh fever; and these three medicines administered in three different kinds of anæmia will restore the wanting iron to the blood and muscles, not because they furnish iron to the economy, but because by restoring the organs to a sound state, they enable them to accomplish their normal functions, in virtue of which they assimilate the iron contained in the food as in the plenitude of health.

Are you not struck by another curious fact? Consequent upon some great mental emotion, or from some other cause, the menses when they had just commenced, are suddenly suppressed, chlorosis often appearing within a few days. For a long time, the malady continues; then, without our being able to discover the causes, there is a profuse menstrual flux, and, after some days, the health seems to be reestablished. Here, the loss of blood, and of its contained iron, is the condition under which the return to health has taken place: in the former case, the loss of blood has been too small, and the iron has disappeared from the blood-globules. You will understand, Gentlemen, that the explanation of these facts is not easy for those who would find in the ferruginous remedy prepared by the apothecary the element necessary for the reparation of the blood.

Perhaps we may render an account of these facts by supposing, that in cases of suppressed menses, there is suppression of an eliminatory function, of an emunctory of morbid principles; whilst with the return of the catamenia, there would be elimination of these same morbid principles. But this double hypothesis will not explain the manner in which iron acts on the blood. And if it be admitted that chlorosis is only a nervous state which produces an immediate effect upon the composition of the blood, ought we not perhaps to see in the preparations of iron only agents which present a modifying influence upon the nervous system, which act on chlorotic anæmia in the same way that mercury, the iodide of potassium, and cinchona act upon syphilitic and paludal anæmia.

LECTURE LXXXVIII.

CIRRHOSIS.

Cirrhosis is not a special product: still less is it atrophy of the red and hypertrophy of the yellow substance of the liver.—It is Chronic and generally Consecutive Phlegmasia.—Cirrhosis in Affections of the Heart, in Alcoholism, Syphilis, and Marsh Fevers.—Slow, progressive Atrophy of all the tissues of the Liver from Strangulation.—Serious Disturbance of the Hepatic Hematosis, and its Response in the Organism.—Cholesteremia.—Cirrhosis, which is a Lesion and not a Malady, adds its evil consequences to the evils belonging to the primitive affection in which it originates.

Gentlemen:—Here are the morbid anatomical preparations taken from a woman, aged 29, who died in the clinical wards from a disease of the heart. Three years previously, she had had a very slight attack of acute articular rheumatism, consisting in pains in the right shoulder, which were of recent date, and unaccompanied by fever. Six months later, she had begun to suffer from palpitations. At the end of a year, her legs became swollen; and at a later date, her abdomen was tumefied.

When admitted to the hospital, this unfortunate woman had most distressing orthopnœa: fine subcrepitant râles were heard in nearly every part of the lungs. No purring thrill was heard over the apex of the heart: there existed, however, a blowing sound, which was not rough, at the apex: this blowing sound, though soft, was intense: its maximum of intensity was at the middle rather than at the apex. There was no venous pulse, and, consequently, no tricuspid insufficiency. I diagnosed contraction of the left auriculo-ventricular orifice and insufficiency of the mitral valve. There was a considerable amount of ascites. The volume of the liver was nearly normal. If, as I suspected, cirrhosis existed, it was but little advanced. There was considerable anasarca: the œdema of the inferior extremities and of the abdominal parietes was enormous. As yet, there was no well marked dilatation of the subcutaneous veins of the abdomen.

Two days after her admission to the hospital, this woman sank under the rapid progress of asphyxia, against which all our treatment was fruitless.

At the autopsy, we found a small heart, without hypertrophy of the left ventricle, and without contraction or insufficiency of the auriculo-ventricular orifice. The ventricular side of the orifice presented a *cul-de-poule* appearance, and the auricular side had the form of a linear slit. The valves were five times their natural thickness: they had the colour and consistence of fibro-cartilage. The aortic orifice was healthy. There was no coagulum in the pulmonary artery.

The lungs, as you see, are remarkably deformed at their summits. In some places, they are shrunk up: one presents two and the other three curious projections, one being inserted within the other like a finger in a glove. One might say, that there is hernia of the lung: in point of fact, there exists a circular depression of the lung caused by retraction of the pleura, which is lined at certain points by false membrane which has become fibrous, and the lung projects at these places where the retraction did not exist. The greater part of the lung is in a state of chronic congestion. The parenchyma is partly carnified, its anatomical condition resembling cirrhosis of the liver.

The liver is normal in volume; and is rather large than small in size: the right lobe is much congested, and very red, having on the red ground, numerous yellowish points as large as the head of a nail, which imparts a granitic appearance, without there being any granulations projecting from the surface. On the contrary, the whole free margin of the organ, and nearly the whole of the small lobe, present a tawny colour and granitic aspect, with projecting granulations, which are the more obviously projecting the nearer they are to the sharp margin. On the inferior surface, you can see depressed, whitish lines, the depression being the result of the retraction of the hypertrophied fibrous tissue—the hypertrophy of the fibrous tissue revealing itself by these furrows.

You see here, Gentlemen, a case of cirrhosis which is not much advanced. The naturally bulky part of the liver—that is to say, the right lobe—is still bulky. It is much congested, and of a bright red colour: the incipient alteration only reveals itself by the yellow colour of the acini. Indeed, in situations in which the organ presents a less degree of thickness, and, where, consequently, there is less predominance of fibrous over parenchymatous tissue, the granular condition is developed. Without it being necessary to go at length into the subject, you can understand the mechanism by which is produced a structural alteration of this nature: as the atrophy of the organ is caused by the retraction of the interstitial fibrous tissue, it is evidently in the situations where this tissue most abounds that atrophy ought to commence, and there also it ought to attain its maximum in the cirrhosed liver. Hence, in all descriptions and pictorial representations of cirrhosis, the sharp edge of the organ, and of its small lobe, are described and figured as being greatly shrunk up. Frerichs of Berlin, to whom we are indebted for a remarkable treatise on diseases of the liver, has mentioned the fact without, however, laying much stress upon it; and in the work to which I refer, you will see a case "in which the liver weighed 2,200 grammes: the right lobe was much swollen, covered with contracted cicatrices, circumscribing tuberosities the size of a hazel-nut, or even as large as a hen's egg, while the left lobe was transformed into a sort of short, tough granulated appendix."[1] Thus, retraction of the interstitial fibrous tissue, strangulation of the parenchyma—and that in the situations where, normally, it is less abundant, were the facts we derived from this autopsy.

But was this really a case of cirrhosis? Is cirrhosis a consequence of cardiac disease? Dr. Frerichs, the justly celebrated German author whose work I have just been quoting, holds that it is not. This is a doctrinal point which we must discuss. Before proceeding to do so, allow me, however, to give you a short history of cirrhosis.

It was Lænnec, as you know, who created the name, *cirrhosis*, deriving it from κέρρος, *russet coloured*, in allusion to the colour of the diseased organ. Dominated by his views in relation to the nature of cancer and tubercle, which he wished to regard as similar in their nature to parasitic organisms, and which he looked on as new products, living their own life at the expense, as well as to the great injury of the organism whence they derive their support, and the destruction of which they ultimately induce—dominated, I say, by these ideas, Lænnec regarded cirrhosis as a special product developed in the liver, there manifesting a particular manner of evolution, the last stage of which is softening.

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[1] Frerichs:—Traité des Maladies du Foie; traduit de l' Allemand par les Docteurs Louis Duménil et I. Pellagot, 2me édition, p. 336. Paris: 1866.

But, long before Lænnec's time, cirrhosis had been observed and described: only, it had often been confounded with tubercle, from the mammellated aspect which it gives to the liver. Morgagni, who employs the expression "tubercles," refers more to the configuration than to the essential nature of the morbid product. He notices the coincidence of ascites with this alteration of the liver. Baillie, in his first article on the disease, gave a summary description of cirrhosis under the designation of "common tubercle of the liver." His description was, to a certain extent, in advance of Morgagni; for he mentions the greater frequency of the malady in men, "probably," he remarks, "because they drink more than women:" he called attention to the corelation of this affection with drunkenness, and to its coexistence with ascites: he also pointed out the diminution in the volume of the blood-vessels, and, after giving a sufficiently good description of the granulations, he insists upon the diminished volume and increased density of the organ.

Baillie, in a second article—entitled "Liver very hard in its substance"—considers this peculiar condition as probably forming the first degree of "common tubercle of the liver." He hazards a pathogenic theory, to the effect, that a new material ("additional matter") is deposited in the parenchyma, and there produces the nodosities. He denies, moreover, that the morbid change is of the nature of scrofula (tubercle) or cancer. Here, you see, is progress.

have stated the views of Lænnec, from the respect I entertain for so great a man, and because it is from him that we date the study of cirrhosis. I pass, without comment, the opinion that there is "atrophy of the red and hypertrophy of the yellow substance of the liver," or rather "atrophy of only one portion of the gland, and hypertrophy of the rest of the organ."

In 1840, Becquerel, though erroneously holding that an albumino-fibrous material was infiltrated into the yellow substance of the liver, and so produced hypertrophy of that substance, this hypertrophy being derived in the first instance from compression, and afterwards from atrophy of the red substance, advanced many very correct ideas, one being, that by far the most common cause of cirrhosis of the liver is acute and frequently recurring congestion of that organ. The liver, under the influence of that congestion receives an abnormal quantity of blood, which is more than sufficient for the biliary secretion. The albumen and fibrine of the blood are deposited, and gradually organized, in the weft of the yellow substance of the liver.[1]

Excepting that it involves a belief in the existence of two substances in the liver, and a belief in the infiltration of the yellow substance, Becquerel's theory is a very near approach to the views now current in medical science.

Since the researches of Rokitansky, Gubler, and particularly of Frerichs, cirrhosis is regarded as the result of the exudation of an organisable blastema into the layers of the interlobular connective tissue. This blastema or plastic substance becomes organized into connective tissue: this newly formed connective tissue becomes fibrous, contracts, and, by its contraction, strangles the secretory parenchyma and the capillary portal vessels. Three consequences follow from this contraction, viz. atrophy of the gland, diminution of the biliary secretion, and ascites. There are many other consequences, but these will come into view as we proceed: at present, I only wish to discuss, so far as necessary, the cause of that exudation. Is inflammation the cause? Is some other morbid process the cause of the exudation?

I have no hesitation in saying, with Frerichs, that cirrhosis originates in chronic inflammation. But I hold, in opposition to the opinion of Frerichs, and in accordance with facts, that this chronic inflammation is very often consecutive to a cardiac affection.

Here, there is a very strong analogy. If the affections of the bronchial tubes and lungs which follow diseases of the heart are not inflammatory, what are they? What is their genesis? In some particular part of that wonderful hydraulic apparatus which we call the heart, a dam exists, and then the blood gradually accumulates above the dam in the afferent vessels. Let us suppose, for example, that there is contraction with insufficiency of the left auriculo-ventricular opening—the auricle empties itself with difficulty, and in an incomplete manner, into the subjacent ventricle, a partial reflux into the pulmonary veins taking place: these veins, again, discharge themselves imperfectly: the result is stasis of the blood in the capillaries originating in these veins, and, as a consequence of this mechanical cause, there is induced hyperemia of the bronchial tubes, and even of the pulmonary parenchyma. But this *unintentional* [*non-intentionelle*] hyperemia—pardon me the expression—this hyperemia which was not originally destined to form the first in a series of morbid changes which has produced inflammation as its ultimate state, is the origin of that inflammation. In the first instance, the surcharged capillaries discharge their excess upon the bronchial mucous membrane: the result is, a flux, or bronchorrhœa, and then, as Læennec says, this phlegmorrhagia becomes chronic bronchitis, so obstinate an affection in persons suffering from disease of the heart. On the other hand, the capillaries allow exudation of the serosity encumbering the posterior lower parts of the lungs: hence we have pulmonary œdema. The blood gradually thickens in consequence of this very exudation, and, whether from the fluid subsequently exuded being increasingly rich in fibrine, or, because in the fluid originally poured out the fibrine predominates in consequence of the evaporation which takes place from the free surface of the bronchial tubes, or finally, whether, because the distended pulmonary tissue becomes more irritable, inflammation always supervenes, and thus we have broncho-pneumonia. The damming up, purely mechanical, has caused, in succession, congestion, flux, dropsy, and, finally, inflammation.

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[1] Becquerel:—*Archives Générales de Médecine.*

Similar phenomena are observed in the kidneys: mechanical congestion is followed by albuminuria: to that succeed infiltration of the secretory canaliculi, desquamation of the tubuli, and finally, that bastard inflammation to which Bright has given his name.

But why should we seek for examples in deep-seated organs? Do we not see with our eyes similar phenomena in dropsical persons, the skin of whose legs is distended by œdema? First of all, there is slight erythema; then, desquamation of the epidermis; and then, the superficial layer of the dermis gives way: vesicles and pustules appear; and sometimes, even more or less of the skin becomes mortified. These are unquestionably phenomena of inflammation, the starting point of which was a purely mechanical congestion of the cutaneous capillaries of the lower extremities.

Who then can have any difficulty in believing, that what takes place in the bronchial tubes, the lungs, the kidneys, and the skin may and must occur in the liver?

The principal objection of Frerichs is, that the embarrassment of the cardiac circulation produces dilatation of the vena cava inferior which gradually extends to the hepatic veins, to the very origin of their roots, the result being atrophy of the cellules. The same author also believes, that the atrophy is the result of compression: the parts supplied by the hepatic veins become flattened, while those supplied by the vena porta protrude in the form of granulations. In other words, the atrophy is limited to those parts of the liver which are next to the dilated capillary vessels. At the same time, the walls of the vessels exposed to abnormal pressure become flattened. In the neighbourhood, here and there, upon the envelope of the gland, there is a production of connective tissue which contributes to giving the liver greater consistence.[1]

Here, you see, is constantly presented the idea of mechanical pressure, never the more dynamic and truer idea of consecutive congestion, exudation, and phlogosis. I cannot too emphatically protest against this view; and my object in quoting the exact text of Frerichs is to show you the point to which one may go, in giving a physical explanation of morbid phenomena. This author has, however, described cirrhosis in a chapter entitled:—"Chronic Inflammation of the Liver."

Cirrhosis, then, is rather a chronic inflammation of the liver than anything else; and it frequently occurs as a secondary affection in cases of disease of the heart, as the result of mechanical causes which I have endeavoured to explain.

Alcoholism is a not less powerful cause of cirrhosis. Being immediately absorbed by the veins of the stomach, without undergoing decomposition, as Mitscherlich has shown, the alcohol enters direct into the portal system of the liver. In this way, it traverses the entire chylopoietic apparatus before it reaches the lungs; and it even copiously bathes the tissue of the lobules. You can understand without my making any detailed statement, what a disastrous influence this powerful irritant must exert upon the secretory parenchyma. The less the alcohol is diluted, the greater is its irritant effect. Upon this point, authors are agreed: cirrhosed liver is called "gin-drinkers' liver" by the English. Frerichs states that on the northern coasts of Germany and England, where the lower classes drink very strong spirituous liquors in excess, cirrhosis is more common than in the inland districts of these countries where the use of wine and beer predominates. In support of this statement, the same author says that he has observed cirrhosis more frequently at Kiel on the shores of the Baltic, than at Göttingen and Breslau.

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[1] FRERICHS:—Traité Pratique des Maladies du Foie. 2nd édition, p. 300, Paris: 1866.

The action of alcohol on the liver may be compared to that of certain irritant substances carried along in the stream of the blood of the vena porta and poured out into the intestines. Budd is disposed to attribute the frequency of cirrhosis in India to the immoderate use of curry and other powerful condiments. With Frerichs, I have very little doubt that the abuse of spices, very strong coffee, and some other similar substances produce hyperemia of the liver, at first transitory, but which by being constantly reproduced, may finally determine the production of cirrhosis.

The action of syphilis occupies a place upon a much less prominent platform. Gentlemen, there is syphilitic inflammation of the liver, just as there is syphilitic inflammation of the testicle. The inflammation is superficial, or it is deep seated. Here, let me bring to your recollection what takes place in syphilitic orchitis.

The affection is sometimes periorchitis, and at other times parenchymatous orchitis. But in every case, it is of the essence of the specific inflammation to attack the connective and fibrous tissue of the organ. Thus in periorchitis, there is inflammation of the tunica albuginea, which becomes thickened, and contracts adhesions to the tunica vaginalis. In parenchymatous orchitis, the inflammation is propagated from the periphery towards the centre: the free part of the testicle is first attacked, and the tunica albuginea is thickened: the inflammation then extends along the vesiculæ seminales and vascular cones to the rete testis, which, as also the epididymis, it seldom attacks. The morbid action consists in increased production or proliferation of the connective tissue into the interstitial tissue situated between the canaliculi. In the early stage of this morbid action, we see this tissue transformed into a soft reddish mass rich in nuclei, which afterwards becomes thickened, assumes a tendinous appearance, separates the canaliculi from one another, and causes them to atrophy, after having determined fatty degeneration of their epithelium. Finally, the vascular cone thus affected gradually hardens, becomes tendinous, and retracts, a depression resembling a cicatrix being formed in that situation. When the depression is so very extensive as to be almost general, the entire testicle diminishes in volume, and its surface is segmented by depressions resembling cicatrices. This alteration of the testicle is a true cirrhosis; and what takes place frequently and easily in the testicle is accomplished by a similar process in the liver of certain syphilitic persons.

Just as we have periorchitis, so have we perihepatitis, an affection of the capsule of Glisson, determining to the surface of the organ a sort of miliary eruption, resembling very small warts; and in the same situation, there is a hard, almost callous thickening of the fibrous capsule, and also adhesions, of very characteristic thickness and solidity, formed with the neighbouring organs. Likewise, as we see in periorchitis, the morbid action propagated to the interior of the organ along the filiform prolongations of the tunica albuginea, so may we also observe the affection advance by slow degrees, following the course of the capsule of Glisson. The result is the formation on the surface of the liver of whitish, radiated wrinkled depressions; and in the same situation, there exists glandular atrophy from real strangulation. You can understand, Gentlemen, that when these depressions (which in respect of tissue are of the nature of cicatrices), penetrate deeply into the organ, and unite with one another, they ultimately strangle the organ by their contraction. This, then, is a special form of cirrhosis. You now see, how syphilis produces this affection of the liver.

Intermittent fever has been mentioned as one of the causes of cirrhosis; and Frerichs has observed three cases in which cirrhosis occurred as the sequel of obstinate intermittent fever. Chronic hyperemia of the organ appears to me to explain, to a certain extent, the development of granular induration.

Finally, it is necessary to distinctly understand, that there are a certain number of cases in which the causes of cirrhosis are entirely unknown.

Gentlemen, I have endeavoured, in this pathogenic grouping which I have now given, to avoid etiological trivialities, by making you be present, as it were, at the genesis of the disease. I have not thought it sufficient to tell you that cirrhosis is observed in cardiac affections, in alcoholism, and in syphilis; but have likewise felt it necessary to inquire with you into the manner in which it is produced.

To sum up:—most of the immediate causes of cirrhosis may be grouped under three heads; viz. 1st, chronic hyperemia of a passive character depending on heart disease or paludal cachexia; 2nd, chronic hyperemia of an active character depending on alcoholic toxæmia; and 3rd, chronic active hyperemia depending on syphilis.

In these three groups of cases, the cirrhosis developes itself from the portal system to the parenchyma, proceeding from the centre towards the periphery, and the affection involves the substance of the liver in deep-seated situations: in the latter case, the disease advances from the fibrous tissue to the glandular structure, from the periphery towards the centre. The cirrhosis is less complete, less generalised throughout the totality of the organ. Virchow, however, correctly remarked, that "the cicatricial indurations of the liver do not necessarily follow the divisions of the vena porta."

Each of these causes of cirrhosis has a corresponding form of special alteration of the liver.

In cirrhosis originating in disease of the heart, the liver only presents innumerable small disseminated granulations on its surface, which is usually, to a slight extent, contracted. There exists a kind of compensation between the contractile tendency of cirrhosis and the expansion-force of the congestion arising from vascular stasis. Consequently, the liver is sometimes increased beyond its normal volume from congestion predominating over contraction; sometimes, its volume remains normal from the existence of an equilibrium of the two forces; and finally, there is sometimes a diminution in its bulk: in every case, however, it is minutely granular.

In alcoholism, the granulations are numerous as well as small: there is great diminution in the volume of the liver, the contractility of the new fibrous tissue not being counterbalanced, as in cirrhosis from cardiac disease, by an antagonising force. In the cirrhosis from alcoholism, then, the liver is very small and finely granular on its surface.

In visceral syphilis, the granulations and the lobules are more voluminous, and less numerous: they are disseminated, sometimes in the form of patches, at other times, they coexist with encysted gummata; and thus we explain Lænnec's cirrhosis in hard patches and cirrhosis in cysts [*en plaques et en kystes*]. The contraction is always less than in the cirrhosis consequent upon alcoholism. In syphilitic cirrhosis, the liver is moderately diminished in volume, and is lobular rather than granular.

I trust that I have shown cirrhosis to be a disease which is not always the same, which is not always identical—which is, on the contrary, nearly always secondary, and dependent upon a protopathic affection—that the lesion differs according as the disease is the result of cardiac affection, alcoholism, or syphilis—and that explains the opposing statements of authors, some of whom have described the lesion as atrophy, and others as hypertrophy. The remarks which I have made upon the mechanism of the different forms of cirrhosis must have led you to perceive why it is, that in heart disease, the cirrhosed liver is more frequently hypertrophied, or at least not reduced in volume, and why it is that the cirrhosed liver of alcoholism is atrophied. You must have also seen why the form of the granulations varies according as the cirrhosis is cardiac, alcoholic, or syphilitic.

I shall not enter into long details regarding the hepatic alterations appreciable to the naked eye, as your classical treatises have made you acquainted with them: it will be sufficient to remind you that the serous envelope is nearly always thickened, and of a whitish grey colour, that it has contracted more or less intimate adhesions with the neighbouring organs, that the liver has acquired increased density, and is sometimes as resistant to the scalpel as leather, that the cut surface is intersected by white tracts interlacing around the granulations which they strangle, and which the light of the branches of the vena porta and biliary canals has notably augmented.

I prefer to discuss some less known peculiarities, in relation to the state of the hepatic cells and other parts of the structure of the liver—peculiarities which enable me to embrace the consideration of a great many of the phenomena of cirrhosis.

First of all, let us see what becomes of the secreting part of the gland—the hepatic cellule. Here, in a special manner, Frerichs will be our guide. The pressure which is exerted upon the biliary canaliculi and the capillary blood-vessels causes great disturbance in the nutrition and function of the organ. The hepatic cellules become disintegrated, and are, for the most part, transformed into a brownish pigment collected in small granulated heaps. Moreover, the bile secreted by the cellules which still remain valid, being able only imperfectly to circulate in the compressed canaliculi, accumulates in the cellules themselves, in the form of fine orange-coloured granules. Every thing, even the blood-globules, becomes changed within the obliterated ramifications of the hepatic veins: in consequence of their stagnation, they become decomposed, and transformed into a substance of a dirty red colour, which, ultimately, is infiltrated into the acini of the liver. It is from this mixture of pigmentary detritus of altered cells, the infiltration of the rest of the parenchyma with biliary pigment, and the colouring matter of the blood globules that the special colour of cirrhosed liver is derived—the colour whence the disease takes its name. Thus, you must perceive the want of truthfulness in the doctrines which attribute cirrhosis to atrophy of the red and hypertrophy of the yellow substance—substances which are mere creations of the imagination of anatomists, being non-existent in the normal state.

I have now to speak of constriction of the capillary vessels which terminate in the portal vein. This is a topic of prime importance in the history of cirrhosis.

This condition presents a primary and purely physical obstacle to the portal circulation in the interior of the liver, so as gradually to produce dilatation of the branches, and then of the trunk of the vena porta vein. In this way, Gentlemen, there is produced slight dilatation of the veins, and particularly of the small veins in the falciform ligament. These small veins emerge from the convex surface of the liver, creep over the inferior surface of the diaphragm, anastomose, in the thickness of the abdominal walls, with the epigastric and internal mammary veins, and then with the subcutaneous veins; and on the other hand, they enter into the transverse groove of the sinus of the vena porta. Thus, there appears under the skin of the abdomen a network of more or less dilated veins, which establishes that which you will allow me to call the *pariètal compensatory circulation,* to distinguish it from another kind of compensating circulation of which, immediately, I shall have to speak to you.

It is not then, as Rokitanski and Bamberger have said, by a restoration of permeability in the umbilical vein, which neither is nor can be more than a permanently impervious fibrous cord, that this compensatory circulation in the abdominal walls is established; but by the dilatation of the small veins normally existing in the thickness of the falciform ligament—veins insignificantly small in the normal state, but which have to perform a part from the time that the intrahepatic circulation of the vena porta is blocked up. However, to call them, as does M. Charles Robin, the "subperitoneal accessory portal veins," is, perhaps, to give them too important a name.[1] "Among the small veins of this group, there is always one," according to M. Robin, "which opens direct into the left branch of the portal sinus, at the point where it is attached to the cord of the umbilical vein. It is specially by this vein, which is destitute of valves, that the blood flows from the liver towards the abdomen, when that organ, being affected by cirrhosis, affords only an insufficient passage. But, as it communicates with the other veins the fluid, which flows into its cavity passes partly into them, so that the entire group shares more or less in its dilatation."[1]

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[1] Robin (Charles):—Report on a Memoir by M. Sappey upon "Un Point d'Anatomie Pathologique relatif à l'Histoire de la Cirrhose." [*Bulletin de l'Académie de Médecine,* 1859, *T. XXII, p.* 944.

In five subjects affected with cirrhosis, which M. Sappey injected, he found, in all of them, this enlargement of the accessory portal veins. It would appear, therefore, as M. Robin justly remarks, that the collateral circulation, which is carried on in consequence of dilatation of normal veins, is more usual than has hitherto been generally supposed. Only, as the reflux sanguineous current generally flows through the epigastric veins, which are deep-seated and anastomose by numerous vessels with the accessory portal veins, it results that the subcutaneous veins dilate more slowly than the deep-seated epigastric veins—that is to say, when they are no longer sufficient for the return of the blood carried by the accessory portal veins: and death sometimes supervenes before they have become sufficiently enlarged.

Dilatation of the superficial veins of the abdominal parietes may be considered, therefore, in the great majority of cases, as a symptom of advanced cirrhosis; but we must not adopt M. Sappey's opinion, "that it ought to be regarded as a somewhat favorable sign, from its removing or diminishing the causes of ascites," because the compensatory circulation is very feeble, and, moreover, from its lessening the causes of ascites, it embarrasses the normal circulation in the abdominal parietes, thereby rapidly producing œdema in that situation. My colleague, M. Monneret, has observed that the abdominal wall is rendered œdematous, and that the dermis itself becomes infiltrated, before the quantity of fluid effused into the peritoneum is sufficient to explain that œdema by ascribing it to distension of the abdominal parietes.

These new conditions under which the circulation is carried on in the subcutaneous abdominal veins sometimes determine the appearance of a double phenomenon which I have pointed out to you in a woman who lately occupied bed 2 of Saint-Bernard's ward. You no doubt remember this woman: her age was about fifty. Veins of great size ramified on her abdominal walls. On applying the finger to any one of these veins, a vibratory thrill was perceived in it, exactly similar to that observed in the jugular veins of anæmic subjects; and which is always an indication of the existence of a vascular blowing, of which this thrilling sensation is the co-relative phenomenon. I was, therefore, convinced that we should hear, on auscultation, a blowing sound in the dilated and vibrating superficial veins. Indeed, along with me, you have been able to ascertain that there existed in this woman a soft vascular murmur perceptible by the stethoscope. It would appear that this fact had been previously pointed out by M. Pégot, and afterwards mentioned by M. Bamberger:[1] but I was ignorant of their researches when I noted the similar observations I had made. To me, it is utterly indifferent who has the merit of priority in this matter; and I have not taken the trouble to connect this unusual development of the abdominal veins with cirrhosis. I asked M. Sappey to have the goodness to come to see this patient, and also to make with me the examination of the body after death. In point of fact, it was in this subject, injected by M. Sappey with his accustomed dexterity, that he discovered the collateral veins of the suspensory ligament: and in this subject also, he was enabled to demonstrate that the compensatory circulation was carried on by the dilated normal veins, and not by the umbilical veins.

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[1] Robin (Charles):—Same Report, p. 945.

Excuse my giving you these anatomical details, which perhaps I have stated at too much length: and bear in mind, that I had to speak to you of a fact which was new or at least little known—the vibrating thrill and the sucurrus in the subcutaneous veins when very much dilated—and that I had likewise to correct the error which attributed this compensatory circulation to the restored patency of the umbilical veins: for these reasons, the details which I have given seemed indispensable. I now revert to the consequences of constriction of the terminal extremities of the portal capillaries.

I have told you that the first of these consequences is the establishment of a parietal compensatory circulation; and the second, the development of a compensatory circulation in the hepatic artery. In the normal state, as you are aware, the terminal capillary branches of the vena porta frequently anastomose with those of the hepatic artery, or in other words, there is anastomosis between the secretory and nutrient circulations of the organ. In the normal condition, this fact can be demonstrated by injections. It can also be shown by injections, that the anastomosis is much greater in the cirrhosed than in the healthy liver. In the cirrhosed liver, from which the hepatic cellules and acini have disappeared, where consequently there are no longer the areoleæ which belong to the acini, where, as a result of the morbid action, the cellules and acini have been replaced by connective tissue of recent formation, we find an entirely new distribution of the capillaries, which have their direction modified, and admit of being injected not only by the vein, but also by the hepatic artery. "Thus it is," says Frerichs, "that new channels of communication are formed between the vena porta and hepatic veins, though their number is limited and inadequate, considering the quantity of blood which traverses the vena porta."[1]

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[1] Robin (Charles):—Same Report, p. 960.

In connection with this fact, Gentlemen, I cannot avoid mentioning another which has been pointed out by my colleague, M. Natalis Guillot, in relation to pulmonary tuberculisation. This able physician, as you know, has shown, in respect of pulmonary tuberculosis, that in proportion to the degree in which the area of the circulation of the pulmonary artery is diminished, the circulation is augmented in the bronchial arteries. The cirrhosed liver, therefore, like the tuberculosed lung, has a new circulation: there is a diminished capacity for portal blood in the one organ, just as, in the other, there is a diminished capacity for the blood of the pulmonary artery: inversely, the capacity for the blood of the hepatic artery, and for the blood of the bronchial arteries, is respectively increased. To express the same fact in different language:—both diseased organs receive less blood for functional, and more for nutritive purposes. Unfortunately, the nutrition is morbid.

The new circulation in the tuberculosed lung is useless for pulmonary hematosis, just as is the compensatory circulation in cirrhosed liver for hepatic hematosis. When the pulmonary vesicules no longer exist, there is a final cessation of the exchange of gases: and when the hepatic cellules have disappeared, secretion of bile and formation of glucose are impossible. There is, in both cases, an arrest of hematopoiesis.

A third consequence of the constriction of the terminal capillaries of the vena porta is a reflux of blood into the spleen, and consequent congestion of that vascular organ. As the spleen undoubtedly plays an active part in the formation of the blood, be it in forming the red globules or the white globules, or in destroying the red globules, we can understand that hematopoiesis is disturbed by stasis and embarrassment in the splenic circulation.

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[1] Frerichs:—Traité Pratique des Maladies du Foie, p. 297, 2me édition; Paris, 1866.

A fourth consequence of the constriction of the portal distributive capillaries is reflux and stasis throughout the originating portal capillaries, that is to say, throughout the entire mucous membrane of the digestive canal. Thus it is, that intestinal absorption is diminished and then suspended: the nutritive materials, the nitrogenous substances, are no longer drawn by these capillaries into the intestinal villi, poured from them into the vena porta, conveyed from them into the liver, to be there elaborated and modified, and then delivered by the vena cava to the heart, to be sent by it to the deepest seated and most remote parts of the organism. You can understand, Gentlemen, that the cessation of this succession of phenomena very directly and very seriously compromises the function of hematopoiesis.

A fifth consequence of embarrassment in the portal circulation of the liver—a consequence more rapid, more easily seen, and consequently better known—is the effect produced on the peritoneum. Vascular stasis in that situation causes dropsy. The ascites is directly proportionate to the duration and gravity of the cirrhosed state of the liver. It is a means provided for the relief of the engorged radicles of the vena porta.

A final consequence, a natural and rationally inferred result of embarrassed portal circulation in the liver is hyperemia of the mucous membrane of the stomach and intestines—with, as a result of this hyperemia, hemorrhages and fluxes. But here, observation and induction are not in accord. Gastro-enterorrhagia is of rare occurrence, as Frerichs himself is obliged to admit: in a certain number of cases, however, it happens, that increased pressure in the capillaries leads to their being torn, so giving a sanguineous coating to the mucous membrane. Fluxes, however, which may occur in the form of gastorrhœa or enterorrhœa, are equally rare: here, consequently, I repeat, observation and induction not being in harmony, we are obliged to combat assertions of a rather hypothetical character by an appeal to facts.

Having stated the consequences of obstruction to the portal circulation in the interior of the liver, I have now to speak of the organic derangement, and functional disturbance which take place consequent upon alteration of the biliary passages and hepatic cellules.

The same cause which produces strangulation of the capillaries of the vena porta, also, in part, strangles and atrophies the radical biliary canals. The principal branches of these canals sometimes participate in the general inflammatory irritation of the gland, presenting, at the same time, increased development of their walls, and thickening of their mucous membrane. Sometimes, also, the walls of the gall-bladder are hypertrophied; and, like the liver itself, have contracted adhesions with the neighbouring organs. The liquid contained in the gall-bladder is tenuous, flowing, of a pale yellow or orange colour, and is evidently a mixture of bile and mucus.

We have seen that many of the hepatic cellules are destroyed: the consequence of this is a great diminution, and sometimes an almost total suppression of the secretion of bile. Thus, the bile ceases to play its part, whatever that may be, in intestinal digestion, which is necessarily very much disturbed. This disturbance is probably the cause of an excessive production of gas in the intestines. At all events, we can see in the disturbance of digestion, in the imperfect elaboration of the alimentary substances entrusted to the intestine, a new and powerful cause of wasting and anæmia. Thus, in cirrhosis, everything seems to concur to produce an altered state of the blood, and destruction of the organism.

The remarks I made to you on the etiology of cirrhosis must have led you to the conclusion that the *symptomatology* of the disease is exceedingly complex and obscure. In fact, of all the symptoms directly referable to it, the most prominent and characteristic is ascites.

The progress of the local affection is generally slow and insidious, no well-marked symptom appearing at its commencement; or, at most, in some very rare cases, we have, as indications of the inflammation, dull pains in the right hypochondrium, and slight increase in the volume of the liver, causing it to extend beyond the false ribs: at the same time, the tongue is loaded, there is anorexia, sometimes accompanied by nausea, vomiting, and a little jaundice. There is sometimes also slight fever, particularly in the evening. These exceptional occurrences are exceedingly transitory.

Progressive diminution of appetite, increasing slowness and great difficulty in digestion, flatulence and constipation, are generally present. The general nutrition is at fault, the patient loses flesh, and his strength fails: from what I have said to you regarding the pathological physiology of cirrhosis, you can see how this must be so. The skin loses its colour, the anæmia becomes more and more evident, and the whole appearance of the patient indicates the existence of that great disturbance of the function of hematosis of which I have had to lay before you the many co-operating causes.

Ascites has already shown itself: it increases slowly, advancing in accordance with the progress of degeneration of the liver. Generally, the abdomen acquires a characteristic appearance from the increased development of the subcutaneous veins. The ramifications of these veins can be seen under the skin, which they raise up: they form a vascular network radiating from the umbilicus up towards the epigastrium, and down towards the inguinal region. Such was the case in our patient of bed 2, Saint-Bernard's ward, in whom the exceedingly dilated veins were the seat of a vibratory thrill, accompanied by a vascular blowing sound perceptible by the stethoscope.

Sometimes, under these circumstances, the abdominal walls and lower extremities become infiltrated. The production of this œdema has been attributed to the compression of the common iliac veins and the vena cava inferior by the effusion into the peritoneal cavity. But is not this œdema partly explained by the disturbance in the circulation in these regions, which is caused by the reflux in the sanguineous current from the vena porta? Opposed to the sanguineous current passing from below upwards by the epigastric veins is the new current proceeding from above downwards from the vena porta. Any obstruction to the circulation in the epigastric veins must be felt in the external iliac and crural veins. A similar cause produces a similar effect upon the venous circulation of the abdominal walls.

Be that as it may, the emaciation of the face, superior extremities, and trunk, form a contrast to the œdematous tumefaction of the inferior extremities.

I shall not expatiate on the local disorders which are associated with the existence of ascites—such, for example, as embarrassed respiration proportionate in degree to the amount of effusion, and a more marked meteorismus consequent upon derangement of the digestion.

Little by little, the general debility and wasting increase, and death comes as the slow result of the progressive exhaustion, or it may occur more rapidly from an intercurrent affection, commonly called a complication, but which is, in my opinion, merely the consequence of the same morbid state which has caused the cirrhosis. Thus, it is not unusual to observe as the terminal phenomenon pulmonary apoplexy, secondary bastard pneumonia, or pleurisy, when the cirrhosis coexists with cardiac disease. In one addicted to alcoholic drinks, the cirrhosis is coexistent with Bright's disease or delirium tremens. Do not be surprised at the frequency of the coincidence of cirrhosis and Bright's disease; both are produced in drunkards by the action of alcohol, and both are results of prolonged passive congestion in persons suffering from disease of the heart. The fact had often been stated; but without the common etiology of the two affections being pointed out. Occasionally, however, real complications occur, such as pulmonary tuberculosis and carcinoma.

But, Gentlemen, perhaps an objection presents itself to your minds similar to that which has often presented itself to my own. Cirrhosis is really chronic atrophy of the liver. You are aware that when the liver ceases to perform its functions, there is a cessation in the metamorphosis of the material, and a consequent accumulation in the blood of substances which, as the result of that metamorphosis, constitute the elements of the bile. It is then that there appears in the urine a number of substances foreign to the organism, such as leucine, tyrosine, and a peculiar extractive matter. Uric acid shows itself only in moderate quantity; and urea, which is the final result of the decomposition of the albuminoid substances, little by little, entirely disappears. How, then, does it happen, that in cases of chronic alcoholia, we have not the disorders of the nervous system, which have been pointed out as occurring in acute atrophy of the liver, that is to say, delirium, convulsions, and coma—or, at least, that their occurrence should be so exceptional as never to have been met with by me, and to have occurred in only two cases mentioned by Frerichs.[1]

Here, it is impossible not to advert to albuminuria, in which affection we sometimes observe the presence of alarming subacute symptoms, and, at other times, the absence of such symptoms. According to the views of modern chemical physicians, uræmia is caused by the accumulation in the blood of urea, which the diseased kidneys are no longer able to eliminate. But if the symptoms of uræmia depend upon this cause, accumulation of urea in the blood, which is entirely physical, and upon adulteration of the blood from the decomposition of that urea into carbonate of ammonia, a consequence entirely physical, it is both a physical and a chemical necessity that the adulteration of the blood should be greater in proportion to the extent and duration of the morbid state of the kidneys. These are exactly the conditions existing in chronic albuminuria. How is it then, that it is precisely in this form of albuminuria that uræmia is most unusual; and that, on the contrary, it is observed in acute albuminuria, that is to say, in the cases in which poisoning of the blood by urea cannot be complete?

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[1] Frerichs:—Traité Pratique des Maladies du Foie, p. 285. Paris, 1866.

Is this an example of tolerance; and must we class these facts with the facts so commonly observed in acute and chronic phthisis? In chronic phthisis, for example, we see great loss of substance. We reduce the field of hematosis to a minimum, and yet the patient continues to live; whereas, in acute phthisis, with much less extensive lesions, and a larger field of hematosis, the patient is asphyxiated. Possibly, in the chronic atrophy of the liver called cirrhosis, and the chronic destruction of the lungs called pulmonary phthisis, there exists a parallelism between the deterioration of the organism and that of the organ, so that the requirements of hepatic and pulmonary hematosis diminish with the diminishing size of the organ. To such an extent is this the case, that the patient may go on living for a long time, though in a very imperfect manner, as the supply is always less than the demand, till the time arrive when the continuance of life ceases to be compatible with an absolutely inadequate production of blood. On the other hand, in acute atrophy of the liver, as in acute phthisis, the organism is abruptly, not gradually, deprived of the sanguineous supply which is indispensable, the disturbance which ensues being so great, that death speedily takes place.

Again, why is it, that cirrhosis, notwithstanding the undoubted atrophy of the liver which it produces, so seldom causes jaundice; whereas, it is one of the principal characteristics of acute atrophy, a characteristic which has conferred on that affection the name of *ictère grave?* Is that explained by the fact that in cirrhosis the atrophy takes place by slow degrees, and that in spite of the destruction of the greater number of hepatic cellules, some of them always remain to secrete the bile; whereas in acute atrophy, the morbid change advances simultaneously and rapidly in all the secreting cellules? Frerichs observed jaundice in seven only of thirty-six cases of cirrhosis, and, except in two of the seven, it was slight. He attributed this slight jaundice to pressure on the radicles of the biliary passages by the connective tissue recently formed around the lobules of the liver. "It is," he says, "the same cause which imparts a yellow colour to the liver itself, and which is the origin of the term cirrhosis."

You are also aware that hemorrhage from all the passages is one of the most remarkable symptoms of *ictère grave*. In fact, so common an occurrence is hemorrhage in acute atrophy, that my colleague, M. Monneret, has described the affection under the name of hemorrhagic jaundice [*ictère hémorrhagique*]; and the hemorrhage is attributed to a fundamental change in the constitution of the blood. Now, hemorrhages are far from being frequent in cirrhosis. I have told you that the hemorrhages which may result from stasis of the blood in the mucous membrane of the digestive canal are by no means of frequent occurrence. As to cutaneous hemorrhages in the form of petechiæ, or as to hemorrhages into the pituitary and buccal membranes, or on the surface of serous membranes, or in the form of cerebral or pulmonary apoplexy, I do not know of one example, if I exclude hemorrhages depending on a disease of the heart, of which the cirrhosis itself is an effect. Frerichs states, however, that they are not so very uncommon. Be that as it may, they are infinitely less frequently met with than in acute alcoholia, although in cirrhosis there exists great atrophy of the liver, and great consequent disturbance of the function of hematosis.

I have still to lay before you certain opinions of a New York professor of physiology—opinions which have a direct bearing on cirrhosis. Dr. Flint has made some very ingenious experiments which have led him to conclude that the liver is in respect of cholesterine what the kidneys are in respect of urea—an organ of elimination and not of production. He considers that cholesterine is an *excrementitial* product formed to a great extent by the *disassimilation* of the brain and nerves; that it is an effete substance [*substance usée*] separated from the blood by the liver; and that it is thrown out into the upper part of the small intestine. In passing along the digestive canal, the cholesterine is transformed into *stercorine*, a ternary compound identical with the *séroline* of Boudet: the stercorine is finally expelled with the fæces. It is not cholesterine then, but stercorine which is found in the fæcal matter.

Here are the proofs given in support of these doctrines:—The blood of the internal jugular vein contains a much larger proportion of cholesterine than is contained in that of the carotid artery, which in fact contains very little: therefore, the blood which returns from the brain is much richer in cholesterine than that which goes to it: consequently, it must be in the brain that the cholesterine is produced. Again, the blood of the femoral vein contains more cholesterine than the blood of the femoral artery: that is to say, that the nerves of the lower extremities have furnished this predominance of cholesterine in the venous blood. Let me remark, however, that I look upon the conclusion arrived at by Dr. Flint as not strictly logical; for it may be quite well alleged that the excess of cholesterine is derived from the muscles, or from all the tissues of the lower extremities, as well as of the nerves. The experiment in which it has been shown that the venous blood of paraplegic limbs contains less cholesterine than that of healthy limbs only proves that the nerves produce the cholesterine, seeing that in paralysed muscles, nutrition unquestionably suffers, and that in them, consequently, disassimilation must necessarily be languid.

On the other hand, the blood of the hepatic veins is, according to Carter's experiments, much less rich in cholesterine than that of the hepatic artery; that is to say, that the blood which comes from the liver contains much less chlolesterine than that which goes to it. To the extent then, that cholesterine is formed by the nervous system, it is taken up by the blood, which throws it off as it traverses the liver.

It is evident, that if this were the case, suspension of the functions of the liver must lead to a cessation of the elimination of the cholesterine, which, by accumulating in the blood, will produce cholesteræmia.

Chemical analysis of the blood of a patient who had been labouring under cirrhosis, and who died in a state of profound stupor, demonstrated to Dr. Flint that there was a large increase in the amount of cholesterine contained in the blood. Becquerel and Rodier also found a large quantity of cholesterine in the blood in an analogous case.

The nervous complications to which Frerichs has directed attention, and of which I have spoken to you, are complications which sometimes supervene at the end of cirrhosis, and which the learned German physician[1] attrributes to alcoholia, that is to say, to suppression of the functions of the liver and a cessation of the production of bile. Dr. Flint attributes the symptoms to cholesteræmia, comparing them to those produced by uræmia, which, as you know, is the result of the accumulation of urea in the blood, consequent upon suspension of the functions of the kidneys.

Dr. Flint says, that the reason why cholesteræmia does not exist in every case of cirrhosis is because the whole of the liver is not disorganised, and that a part of the organ suffices to eliminate the cholesterine, just as, in cases of degeneration or ablation of one kidney, the other continues to eliminate urea.

I must add, that the very interesting experiments of Dr. Oré (of Bordeaux) appear to support the views of the New York professor. In fact, it would appear from the experiments of Dr. Oré, that the bile is secreted from the blood of the hepatic artery, and not from that of the vena porta. We know, on the one hand, that the portal blood contains nitrogenous matters derived from the digestive canal, and very little cholesterine; and on the other, we know, from the analyses made by Carter, that the blood of the hepatic artery contains a large quantity of cholesterine.

Gentlemen, I have thought it right to place before you this ingenious theory. It is specially important to take every opportunity of bringing into comparison the results of clinical observation and experimental physiology. In respect of cirrhosis, I have also thought it incumbent on me to inquire, along with you, into the possible mechanism by which this redoubtable lesion of an organ so important as the liver inflicts such irreparable damage on the economy.

Were I to wish now to embrace in one general view the entire pathological mechanism of cirrhosis, I should perceive, that it consists of a concatenation of phenomena which succeed one another in a necessary order, because they originate the one from the other. In chronological order, the phenomena are:—exudation of an organisable blastema into the interlobular connective tissue—organisation of this blastema, which becomes fibrous—gradual retraction of this fibrous tissue, which both strangles the secretory parenchyma and compresses the divisions of the vena porta. Thence, as a twofold consequence, we have consecutive atrophy of the liver, and gradually increasing embarrassment in the portal circulation.

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[1] Robin (Charles):—*Journal de l'Anatomie et de la Pathologie,* for September, 1864.

But we have seen, that as soon as the portal circulation is impeded, the secretion of bile and of glucose becomes less and less; and that in respect of nutrition, the alimentary materials taken up by the radicles of the mesenteric veins ramifying on the mucous membrane of the intestine, enter with increasing difficulty the vena cava, thence to pass into the general circulation.

The organism, therefore, is not only threatened on account of the hematosis being incomplete in consequence of the ever diminishing secretion of bile and glucose, but also on account of nutrition being directly interfered with in consequence of the inadequacy of the amount of assimilable materials floated along the vena porta, then along the vena cava, and finally propelled by the heart to the remotest recesses of the organism, to carry thither the elements of reparation.

We have already seen the direct consequences to the economy of a morbid state of the liver. Nor is that all: we have seen that cirrhosis is generally a secondary affection occurring in the course of, and as a consequence of, a previous malady which contributes a large share to the deterioration of the organism.

Thus, in the subject affected by alcoholism, there is diarrhœa with concurrent chronic gastritis of sometimes an ulcerous character, the alcohol exerting its direct deleterious action on the mucous membrane of the stomach prior to its irritating the liver. The dyspepsia resulting from the malady of the stomach disturbs nutrition at its very source, and becomes additional to the dyspepsia proceeding from the cirrhosis. The series of lesions and functional disorders which belong to chronic alcoholism, are also often superadded: to this category belong fatty degeneration of the heart, atheromatous degeneration of the arteries, atrophy of the brain from true cirrhosis, chronic bronchitis, even pulmonary tuberculosis, granular disease of the kidneys, all of which, I need not say, exercise an evil influence on the entire organism.

In one who is the subject of heart disease, the position is also very serious. In such an individual, the vascular stasis which interferes with hepatic hematosis has already for a long time compromised pulmonary hematosis: in point of fact, he is affected with pulmonary congestion, chronic bronchitis, or bronchorrhœa; and to the debility resulting from the progressive diminution in the pulmonary hematosis, there is added that which is the natural consequence of the loss arising from abnormal bronchial secretion. On the other hand, the renal depuration of the blood is generally imperfect, in consequence of the kidneys being usually in a congested state, and often from the albuminuria caused by the congestion, in which case, the situation of the patient is still more aggravated.

In the syphilitic subject, the poisoning is general: when the virus is so deeply rooted, and the affection so inveterate as to have become visceral, there is not only cirrhosis of the liver but also a structural change in the kidneys and spleen—often also in the lungs. The kidneys frequently undergo amyloid degeneration, and the result of this is albuminuria. Here, everything concurs to produce a morbid change in the blood: the liver is an organ both of hematosis and of depuration: the kidneys serve only as depurators.

The chances of the clinical wards have not enabled me to show you the lesions which belong to syphilitic cirrhosis. To give you a sufficiently exact idea of the nature and extent of the lesions, I cannot do better than quote, in an abridged form, a typical case from the work of Frerichs.

His thirty-ninth case[1] is the history of a woman, thirty-six years of age, who had suffered on several occasions from primary and secondary syphilis. Two years before admission to the hospital, she had been treated for albuminuria complicated with anasarca, from which she was easily relieved. She came into hospital with all the symptoms of a profuse pleuritic effusion of the right side. She died fifteen days afterwards, having had frequent nausea and vomiting, white serous stools, and delirium for a short time.

At the autopsy, in addition to the effusion into the right pleura, a multiplicity of lesions were found. The mucous membrane of the small intestine presented an intensely injected appearance; and its solitary glands were prominent. The large intestine was also injected. The pancreas was hard; and the mesenteric glands had in part become calcareous. The spleen was large, hard, and lardaceous; and, from the presence of amyloid granules, had a brilliant aspect. The kidneys were bulky: their parenchyma was in part hard and lardaceous, and in part friable, with yellow infiltration. Everywhere, there were intimate adhesions between the liver and diaphragm: *the left lobe was completely atrophied,* and was almost indistinguishable from the diaphragm: the right lobe presented, on its convex surface, and on its upper margin, deep cicatricial contractions which circumscribed portions resembling lobes of the size of hazel-nuts. The parenchyma was everywhere knotty, very hard, and of a shining reddish-brown appearance. The bile was thick and mucous, nearly gelatinous, and of a dull colour: it deposited a large quantity of colouring matter, and was without albumen.

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[1] Frerichs:—Op. cit., p. 333.

You see that in this case, the patient presented simultaneously amyloid degeneration of the liver, spleen, and kidneys. In other words, there coexisted disturbance of the function which presides over the formation of the blood by the liver and the spleen, and disturbance of the function by the exercise of which the blood is depurated by the kidneys. It appears then equally incorrect and irrational to consider that this woman succumbed solely from her hepatic affection: it is likewise seen, how difficult it would be to describe the cirrhosis, leaving ascites out of view, as a separate isolated disease, and to say, how much of the complex symptoms in this case were positively due to the hepatic lesion. In point of fact, the woman died from visceral syphilis presenting everywhere the numerous lesions and disorders which belong to this disease, and not only from the liver having become cirrhosed.

When you find granular degeneration of the kidneys described in your treatises on pathology among the complications of cirrhosis, you are inclined to say that an unrecognised general disease is being discussed, and that the effect has been mistaken for the cause, or rather, that there has been a failure to detect the disease on which a series of lesions depended. That is the conclusion to which an excess of pathological anatomy must inevitably lead.

I cannot too often repeat, that cirrhosis, in an immense majority of cases, is *not an isolated disease*, is not, in fact, a disease at all, but a secondary lesion, connected by a relation of causality with other lesions of similar origin, such as those arising from cardiac, paludal, syphilitic, and alcoholic affections; and that it is only by adopting an artificial basis of description, unsanctioned by a correct interpretation of facts, that it has come to be described as a distinct disease, with precisely defined characteristics.

Were cirrhosis regarded, as I regard it, from an etiological point of view, and not from an anatomical point of view—were the lesion looked upon, not as made but as being made—were the idea followed of the action of the general morbific cause we would be present, so to speak, at the genesis of the complications. We would then understand, for example, how, in consequence of the alcoholic poison exerting its irritant action in succession on the stomach, liver, and kidneys, there often occurs simultaneously thickening and ulceration of the gastric mucous membrane, cirrhosis of the liver and Bright's disease of the kidney—how the syphilitic poison acting on the organs of hematopoiesis, produces simultaneously amyloid degeneration of the spleen and kidneys with cirrhosis of the liver—and, finally, how the generality of cause engenders multiplicity of lesion.

The remarks I have now made show you how difficult is the diagnosis of cirrhosis. In the totality of symptoms depending on the general affection, cirrhosis hardly has any other signs which belong to it, except ascites and enlargement of the subcutaneous abdominal veins. For we must beware of supposing that the venous murmur of which I spoke to you is constantly present.

It is with chronic peritonitis, and particularly with tuberculous peritonitis, that the ascites due to cirrhosis may be confounded. But, generally, tuberculous peritonitis coexists, in the adult, with pulmonary tuberculosis; and then, the signs peculiar to the latter affection enable us to recognise the tuberculous origin of the peritoneal effusion. There are, moreover, in tuberculous peritonitis abdominal pains, whereas they do not occur in the ascites arising from cirrhosis. Cases perfectly inexplicable do, however, occur; and the following is an account of one of that description.

A man, forty-seven years of age, was admitted to the clinical wards on the 3rd March, 1864. He had suffered from difficulty of breathing for five months; and so much had his health declined, that he was no longer able to do his usual work. In fact, there was extensive pleuritic effusion on the right side. The pleurisy, on its first declaring itself had been treated by a blister which was three times repeated without any obvious beneficial effect. There was absolute dulness extending from the spine of the scapula behind to the second rib in front. In that situation, no respiratory murmur was audible. The liver was squeezed down as low as the umbilicus. From the great extent of this effusion, I resolved that paracentesis of the chest should be performed by Dr. Peter, my *chef de clinique*. By the operation, 2,300 grammes of serosity were withdrawn: the dyspnœa ceased quickly, and did not return: the patient, however, remained pale, and recovered his strength slowly. Although he had no cough, and although I could detect no abnormal sound at all attributable to pulmonary tuberculosis, yet as his pleurisy was on the right side, had assumed the latent form, had advanced in a chronic manner, and finally, as the fingers were hippocratic, I concluded that the man was tuberculous. My suspicions were in part confirmed, when the patient, who had gone out at his own request, asked, on the 25th April, to be readmitted. He was then thinner and weaker than when he left us; and he had considerable effusion into the abdomen. This effusion had been preceded by wandering abdominal pains. I had no hesitation in concluding that the dropsy was caused by tuberculous peritonitis. No conclusion, apparently, could be more rational. The form and progress of the preceding pleurisy, and the pains which had preceded the peritoneal effusion, seemed to support the view of the simultaneous existence of tuberculosis of the pleura and peritoneum. There was, nevertheless, a speciality in the case which caused me to hesitate, and kept my mind in suspense: it was the absence of any pulmonary sounds peculiar to tuberculosis.

This patient remained six weeks in our wards, becoming feebler day by day, having hectic fever, vomiting nearly everything he ate during the three latter weeks of his life, and suffering slightly from diarrhœa. At last, he died in a state of marasmus.

At the autopsy, we found traces of chronic pleurisy of the right side, with close adhesions between lung and pleura. There was about half a litre of lemon-coloured serosity in that side of the chest; and in the left side, there was nearly a litre of similar effusion. Thus, there were the signs of chronic bilateral pleurisy: and yet we could not find any tubercular deposit in the lungs. Nor was there any tubercle in the peritoneum, which, throughout its whole extent, was exceedingly injected. The intestinal convolutions were, here and there, lined with false membrane, which was thin and soft. The peritoneal effusion was copious, and of a dirty colour. There, therefore, existed chronic peritonitis: there was also a morbid condition which we did not expect to find—cirrhosis of the liver. The liver was small and indurated: it presented, externally, a fine granulated appearance: the granulations were as large as millet seeds, or, at most, as large as hemp seeds: white lines seamed the surface of the liver, and in the situation of the majority of the lines, there were slight depressions, some of which were linear, while others were broader: they all corresponded with the trabeculæ of the capsule of Glisson. A section of the liver presented a fine granulated appearance.

That is, I think, the form of cirrhosis which is associated with chronic alcoholism. It looks like a raking of the organ, which does not present any projections of the size of hazel nuts, such as are found in syphilitic cirrhosis: none of the granulations are larger than hemp seed: there is an almost general thickening of the capsule of Glisson.

The mucous membrane of the stomach was enormously injected: in some parts, it was of a slaty colour, in others, it was bright red; and this latter was the appearance it presented in the neighbourhood of the pylorus. The mucous membrane of the small intestine, particularly in its lower portion, was much injected: there were a certain number of oval ulcerations on the valvulæ conniventes, and some circular ulcerations in the situation of the separate follicules. There was no trace of tubercular deposit in any part of the intestinal canal.

We ascertained, as the result of careful inquiry, that the man had been an habitual drinker of alcoholic stimulants. Appearances, notwithstanding, he was not tuberculous; and we must ascribe his gastro-enteritis to alcoholism. The gastro-enteritis probably caused, by propagation, the peritonitis; for the peritoneum presented the greatest appearance of injection in the situation of the intestinal ulcerations and in that of the vascularity of the mucous membrane of the stomach. The peritoneal effusion, therefore, was due both to the peritonitis and the cirrhosis. As to the latter, there was necessarily misconception, deceived as one had previously been by the anterior pleurisy, and signs of peritonitis—pleurisy and peritonitis which it was much more truth-like to ascribe to the tubercular diathesis.

In fact, cirrhosis ought to be suspected when we have ascites appearing in a cachectic drunkard, or in a syphilitic subject who has reached the period when tertiary symptoms occur: we ought also to suspect its existence in a person affected with cardiac disease, when the ascites is to a much greater degree than the œdema of the inferior extremities, particularly if there be dyspepsia with marked wasting of the face and superior extremities. Finally, there is reason to believe that cirrhosis exists when there is ascites without our being able to discover any material obstacle in the course of the vena porta.

Having told you how cirrhosis, a lesion secondary to a more general affection, adds its share of gravity to a condition already very formidable—having explained to you how, by impeding hepatic hematosis and drying up the very sources of assimilation, it assails life in a doubly dangerous manner—it would be wasting time to insist upon the gravity of the *prognosis*.

In that gravity, however, there are different degrees, according to the period of the lesion and the nature of the affection on which the cirrhosis depends. Thus, for example, incipient cirrhosis is evidently less grave than cirrhosis in its last stage; and cirrhosis depending on paludal toxæmia is less grave than cirrhosis arising from an affection of the heart, which, again, does not become so rapidly formidable as cirrhosis originating in visceral syphilis or alcoholic poisoning.

What I say in respect of prognosis, is applicable also to *treatment*. That is one of the reasons which led me to enter so fully into the etiology and evolution of cirrhosis. Therapeutics, in fact, avail only when the lesion is incipient: when it is confirmed, treatment is of no use.

The endeavours, therefore, of the physician must be directed to prevent cirrhosis, or to impede its progress. When, therefore, in a person who has an affection of the heart, you ascertain that there is enlargement of the liver, local pain, and a slight icteric color, you must set yourself to subdue this manifest congestion of the liver. But as this congestion is connected with embarrassment of the circulation, the means to be adopted for diminishing the general vascular tension and the consecutive visceral congestion are:—regulating the circulation by digitalis, slightly stimulating the system by the use of a little coffee, which acts as a diuretic as well as a stimulant.

Again, when in a drunkard, disturbance of the function of digestion leads you to apprehend the imminence of cirrhosis, you must advise the patient at once to renounce his bad habits, recommending him at the same time to make use of aliments but little stimulating, and easily assimilated.

In cases of syphilitic and paludal cirrhosis, simultaneously combat the specificity of the cause by a specific medication, and the debility of the system by general tonic treatment. You will treat the lesion of the liver, with its immediate consequences, viz., anorexia, dyspepsia, ascites, &c., by an appropriate medication, which will vary with the varying indications, but which, too often, can only be palliative.

I trust that I have now shown you that the history of cirrhosis is, so to speak, only an episode in the history (in other respects very complex) of cardiac, syphilitic, alcoholic, or paludal cachexia:

but then, it is an important episode. It is, let me say, one of the terms in a morbid series embracing all the organs, which commences with congestion, continues by phlogosis, and concludes in cachexia. Once the minute mechanism of cirrhosis is understood, there is little ground for hoping much from therapeutics. Art is necessarily powerless, when the lesion is irremediable. This fact I have already sufficiently impressed upon you.

LECTURE LXXXIX.

ADDISON'S DISEASE.

A Special Disease.—A Peculiar Kind of Anæmia, generally associated with an Affection of the Supra-renal Capsules.—A Few Words regarding the Supra-renal Capsules.—Symptoms of Addison's Disease.—Consequences of the Anæmia.—Peculiar Dingy Color of the Skin.—Difficulty of Diagnosis.—Treatment.

GENTLEMEN:—To testify the gratitude which Art and Science owe to eminent men, perhaps also with a view to avoid the creation of new words, physicians—who, speaking generally, are slow to render justice to their brethren—have given to certain diseases the names of their discoverers, or the names of the individuals who first gave a complete description of them. For example, under the name of "Pott's Disease" we know a disease of the vertebral column characterised by caries of the vertebræ, with or without tubercular deposit, giving rise to abscesses from congestion and usually leading to spinal deformity, sometimes to paraplegia. In the same way, by "Bright's Disease," we mean chronic albuminuria, an affection which the celebrated London physician studied and described better than it had been previously studied or described by any one. In the same way, insufficiency of the sigmoid valves of the aorta is still called "Corrigan's Disease." And it would on the same principle be an act of justice to give the name of "Bouillaud's Disease" to endocarditis, an affection almost quite unknown, till the illustrious Professor of the Hôpital de la Charité directed to it the attention of the medical world, by writing a history of it so complete as to leave nothing to be added by others.

In obedience to a similar feeling of equity, I propose to-day to designate the disease of which the individual occupying bed 5 Saint-Agnes's ward presents a remarkable example, by the name of the

English physician who discovered it. The physician to whom I refer is Dr. Addison, the fellow-laborer of Bright, the Dean of the Medical School attached to Guy's Hospital, London, a man who has long been known among us by his valuable scientific works. I propose then to give the name of "Addison's Disease" to that singular cachexia which is specially characterised by the decoloration, or rather the peculiar coloration—the bronzed tint—of the skin, which obtained for the malady the name under which Addison described it, viz. *bronzed disease.*

In the course of his practice, Dr. Addison had long been struck by meeting with certain forms of general anæmia which were neither attributable to excessive antecedent hemorrhages nor to profuse or long continued intestinal fluxes, and could not by symptoms be connected with any diathesic state or marsh miasmatic affection—forms of general anæmia, in fact, which appeared to supervene under the influence of non-recognizable causes. In studying them, he distinguished from the rest one which was, besides the debility and languor of the patient, characterised by that bronzed hue of the integuments of which I have been speaking, and which is most strikingly apparent on the skin of the hands, penis, scrotum, groin, and axilla.

Dr. Addison having observed that in every case the malady followed the same course, and invariably terminated in death, made very careful autopsies with a view to obtain the greatest possible amount of information regarding the nature of the disease. From the minute manner in which he was in the habit of directing his inquiries into morbid states of the kidney, he discovered that in this singular form of anæmia, the supra-renal capsules were often diseased. The coincidence of a morbid change in an organ, the function assigned to which in the economy had till then been insignificant, the coincidence of an anatomical lesion apparently so insignificant with so serious a general condition of the economy, soon put him on the right track. From the date of his first case, he thought that he had found the explanation he was seeking.

In 1855, Addison published the results of his observations in a monograph containing the history of eleven cases, which, although not all equally decisive, enabled him to conclude, that the *bronzed disease* is associated with a lesion of the supra-renal capsules, without assuming, however, that this lesion (whatever might be its nature) was the cause of the disease, any more than the alteration which takes place in Peyer's glands is the cause of typhoid fever, pustules the cause of small-pox, or, engorgement of the spleen the cause of paludal intermittent fever.[1] In all these examples, the organic lesion is only a characteristic part of the disease, distinguishing it from other morbid species of a similar kind.

The profession having thus been put on the alert in this inquiry, similar cases were, during the same year, recorded by the English medical press; and in France, my friend, Dr. Lasègue, made known Dr. Addison's researches, by publishing an analysis of them.[2]

With the case under notice in our wards, with that example so to speak, under our eyes, it is my desire to-day to give you an idea of Addison's Disease, following the description of the English physician, as reproduced by my learned colleague of the Hôpital Necker.

The following is a summary of our patient's case. His age is thirty. He was coachman to the Minister of the Interior. His occupation, although certainly an arduous one, was not so relatively, because he was well paid, well fed, and well clothed: he had no drawback, except insufficient sleep; and, in fact, only complained of inability to obtain his necessary amount of rest at night. When I saw him, however, at the date of his admission to the hospital, he was under the impression that he had been losing flesh for the preceding four or five months, in such a way as to alarm both himself and his family. For three months, he had observed that his hands retained a dark hue, however much care he bestowed on washing them; that his face was assuming the bistre, smoked aspect of a mulattoe's skin; that the skin of the inside of his lips had a hue reminding one of the interior of the mouth of certain dogs; and finally, that the dark colour was making its appearance on different parts of his body, and was in no degree amenable to the prolonged use of baths.

I verified the change of color which I have now described. The patient's nails had that remarkably white appearance met with in anæmic persons, proving that the dark hue of the hands with which they contrasted was not the result of any neglect of cleanliness. The dark hue was found on the skin of the penis, scrotum, groins, and arms. The areola, however, of the nipple, which, in similar cases, often acquires a deep hue, as in pregnant women, had its natural colour.

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[1] ADDISON:—On the Constitutional and Local Effects of Disease of the Supra-Renal Capsules: London, 1855.

[2] LASÉGUE:—*Archives Générales de Médecine* for March, 1856.

The man stated that his legs bent under him, and that he had lost so much strength as to be unable to accomplish the small amount of walking required of him. A blowing sound, evidently anæmic, was heard on auscultating the heart and vessels of the neck. On examining the different organs of respiration and digestion, the liver and spleen, no organic lesion was discovered; and the functions of these organs seemed to be performed with perfect regularity. The urine—analysed on several occasions—contained neither albumen nor glucose. Except great debility, and the peculiar colour of the skin, the only morbid symptoms complained of by the patient were pains resembling those of rheumatism, which he said he felt, from time to time deep-seated in the sides. These pains ceased quickly, returned without any appreciable cause, and were not fixed in any one place. I attached all the more importance to this point, as the patient presented the symptoms characteristic of the bronzed disease. I endeavoured to discover whether there were any special symptoms indicating an affection of the supra-renal capsules.

Although the patient, who rose every day, walked about the wards, ate and drank very much like a man in health—except, that, like chlorotic women and some patients suffering from cancer, he had a disgust for animal food—was in the full enjoyment of his intellectual powers, and, except anæmia, presented nothing abnormal save the bronzing of the skin, from that symptom I at once formed a very unfavorable prognosis.

It certainly was not my personal experience which led me to this conclusion, for the case was only the second of the kind which I had met with. My first case was that of a young man of twenty-seven. Consequent upon the effects of cold, from discontinuing the use of a flannel belt which he had been in the habit of wearing, he was seized with fever and pains in the lumbar region. He soon fell into a state of great debility, the slightest muscular exertion causing breathlessness and palpitation. The skin looked like that of a mulatto, and, in some places, was quite black: the black hue was seen in old cicatrices, and also on the hands, the nails of which had a variegated appearance, if I may use the expression; that is to say, they were longitudinally streaked with brown bands. The mucous membrane of the lips, gums, and tongue presented similar brown streaks, which contrasted in a remarkable manner with the natural colour of the rest of the mouth.

I lost sight of this young man; so that the case did not afford me much instruction. Consequently, my very unfavorable prognosis in the case of our patient in Saint-Agnes's ward was not based on my personal experience, but on the statements of Dr. Addison and the other English physicians who had spoken of the bronzed disease. I knew that of fifteen patients whose cases had been published, all had died. With such recorded mortality before me, there was, it must be admitted, a strong presumption, that no better fate would attend cases occurring in my practice.

The result too truly realised my fears; and the fatal termination of the case occurred even sooner than I expected. The patient was suddenly seized with a profuse diarrhœa, having as many as eight or ten stools in the twenty-four hours; and the whole body became cold, without, however, there being anything to suggest the existence of cholera in the special appearance of the stools, suppression of urine, or extinction of voice. Delirium supervened: debility made rapid progrees: and on the fourth day from the commencement of these symptoms, death took place.

On opening the body, we found no lesion of lungs, heart, or intestines, sufficient to explain the cause of death. The kidneys, which were examined by me and Dr. Brown-Séquard, presented no morbid appearances, except slight hypertrophy, some tubercular deposit, and some fibrous filaments. But, in the *supra-renal capsules,* there were numerous masses of tubercle; and the left capsule (which was considerably enlarged) was almost entirely transformed into that heteromorphous product.

In the apex of one of the lungs, there was a small mass of tubercular deposit of the size of a lentil. Neither the bronchial nor mesenteric glands contained any tubercle.

The blood, which was examined by Professor Charles Robin, presented no other alterations than those of ordinary anæmia.

Here, then, is an individual, who when still a young man, is suddenly attacked by a cachectic disease which is inexplicable by existing pathological knowledge. The bronzed color of the skin, and the disseminated black patches were, during life, characteristic of the disease described by Dr. Addison; and after death, the only noteworthy lesions we found were the morbid changes in the supra-renal capsules which he has described.

If you will now bear in mind the symptoms of which our patient complained, and those which we ourselves observed in him, you will find that they present a picture identical with that which Dr. Addison has drawn; and which I cannot do better than reproduce.

But before proceeding to do so, let me say a few words regarding the supra-renal capsules. So little information have anatomy and physiology afforded in relation to these organs, that they have hardly engaged serious attention. It was observed, that they were susceptible, like other organs, of undergoing serious structural changes, apoplectiform hemorrhages, tuberculous or cancerous transformations; and that cysts might be developed in their substance. In 1837, Dr. Rayer published a considerable number of cases of this description; but he concluded his work with the discouraging avowal that, up to the date at which he wrote, the study of the morbid changes of the supra-renal capsules offered so little that was interesting that it might, without detriment, be neglected by pathologists; and that it had not thrown any light upon the functions of these organs.[1]

Nevertheless, their richness in vessels and nerves, and the constancy of the existence of the supra-renal capsules, gave reason for believing that their functions were of some importance—just as we suppose to be the case with the spleen, which likewise plays an unknown part in the economy. But there was not an absolute lack of hypotheses: some physiologists looked on the supra-renal capsules as forming part of the urinopoietic system, and others supposed that there was some connection between them and the genital organs.

From the profusion of blood-vessels in the supra-renal capsules, it has been supposed that they perform an unascertained part in hematosis, analogous, probably, to the function of the spleen, or thymus. A remark made during last century, to the effect, that the supra-renal capsules are larger in the negro than in the white race, suggested that they were in some way related to the pigmentary secretion. Bergmann, in his inaugural dissertation, held that they were nervous ganglia. He based this hypothesis upon the anatomical structure of the organs, and on the observations of his father the celebrated alienist of Hildesheim, who maintained, with Jacobson, that they are often found to have undergone morbid changes in diseases of the brain and spinal marrow.

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[1] RAYER:—Recherches Anatomico-pathologiques sur les Capsules Surrénales. [*L'Epérience* for 1837.]

The recent researches of Dr. Brown-Séquard, and the discovery of Dr. Addison, may accelerate the solution of the question.

Let me state the principal facts enunciated by Brown-Séquard. He says, that the supra-renal capsules are very sensitive. In opposition to the hitherto received opinion, that they are very large in the fœtus, and become atrophied after birth, he holds that they increase in weight and volume from birth up to adult age, so that they cannot be looked upon as the remains of an organ of fœtal life.

Extirpation of both supra-renal capsules kills animals as certainly and as rapidly as extirpation of both kidneys. Brown-Séquard performed the experiment upon sixty animals: he states that eleven hours was the average duration of life after the extirpation. When only one supra-renal capsule was removed, the animal did not survive more than seventeen hours. In none of these cases could death be attributed to hemorrhage, peritonitis, lesion of the kidneys, liver, or other important neighbouring organ.

It was observed that when both supra-renal capsules were extirpated, pretty nearly the same series of phenomena was produced; viz. in the first instance, acceleration of the breathing, which soon, however, became slow, jerking, and irregular—increased rapidity in the pulsations of the heart—diminished temperature—and, on the approach of death, nervous phenomena, such as vertigo, convulsions and coma.

This, Gentlemen, leads me to ask whether this profuse diarrhœal flux in our patient was a phenomenon of this description.

Extirpation of only one capsule produces the same symptoms, but more slowly, and not till a period has elapsed during which the animal seems to be recovering: when there are convulsions, they occur on the side on which the extirpation has been performed, the animal turning round like a screw on its own axis, as is observed after section of one of the middle peduncles of the cerebellum: the direction of this rotatory movement is from the side operated on to the other side.

In Paris, an epidemic prevails among rabbits, which is characterised by inflammation of the supra-renal capsules—*an inflammation which produces the same effect as extirpation of these organs.* The blood of rabbits infected with this disease, when injected into other rabbits induces affections similar to those which result from the extirpation and inflammation of the supra-renal capsules.

Wounds of the spinal marrow occasion hyperæmia of the supra-renal capsules, a fact established by Brown-Séquard in 1851: from this results hypertrophy or intense inflammation, under which, in a short time, the rabbits succumb.

From the facts now adduced, we must conclude, with the physiologist from whom I have taken all the details, that the supra-renal capsules are organs essential to life, that their extirpation, alteration of structure, or destruction, influence the economy, either by arresting the functions of these organs as hematopoietic glands, or by inducing irritation of the nervous system.

Brown-Séquard, after demonstrating the analogies and differences between the results of ablation of the supra-renal capsules, the pigmentary disease which he had so frequently observed in rabbits, and Addison's Disease, terminates his memoir by a study of the functions of the supra-renal capsules, concluding that their probable function is to modify a certain substance destined to be transformed into pigment, and to modify it in such a manner as to prevent this transformation from taking place.[1] In relation to that subject he refers to Professor Vulpian's experiments.[2]

Having said this much regarding the supra-renal capsules—the bearing of which you perceive—we have now to inquire:—*What are the symptoms of Addison's Disease?*

The malady begins slowly; and its existence is not at first perceived. The patient has difficulty in fixing with precision the date at which he experienced its earliest symptoms. Its first manifestations are general discomfort, an enfeebling of the physical and moral faculties, and a state of real languor. The arterial pulsations are small and feeble; or the pulse is full, soft, and easily compressed. The appetite is capricious, the patient (like the man in Saint-Agnes's ward), showing repugnance to animal food, or a diminished appetite. At first, digestion proceeds in a normal manner: at a later stage, this function is disturbed by intractable vomiting. This symptom is accompanied by pain, or at least by a painful sensation, in the epigastric region. The patient wastes away; and yet the most minute examination fails to discover any sign of organic change sufficient to account for the great disturbance of health and extreme anæmia which exist. Finally, there is a state of extreme debility, to which, very properly, attention has been directed by a distinguished hospital physician, Dr. Siredey: this condition is made very evident by dynamometric examination.

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[1] BROWN-SÉQUARD:—*Journal de Physiologie;* 1858.

[2] VULPIAN:—*Comptes Rendus de l'Académie des Sciences;* 1856.

However, the characteristic colour of the skin, to which the patient himself directs the physician's attention, or which has at least attracted the notice of those about him, supplies a pathognomonic element of diagnosis. This altogether special brown colour occupies the entire surface of the body, and is nowhere more marked than on the skin of the face, neck, superior extremities (particularly on the hands, where it forms a remarkable contrast to the usually colourless nails), on the scrotum, axilla, and around the umbilicus. In these situations, the colour shows itself in dark patches, varying from a clear brown or bistre to a bronze hue: the mucous membranes present similar appearances. Both the man of Saint-Agnes's ward, and the young man whom I saw in my private consulting-room were thus affected: the one had the lips, and the other had the lips, gums, and tongue marbled with black patches, which, to use a comparison I have already employed, recalled the appearance of the inside of the mouth of certain dogs. Very recently, Dr. Hérard caused notice to be taken of a similar coloration of the lips and gums, of a woman whom he showed to the *Société Médicale des Hôpitaux*.[1] It is not the mucous membranes only which show this remarkable coloration: Dr. Addison states, that in one subject, he observed similar brown patches on the peritoneum.

These patches are constituted by deposits of pigment irregularly accumulated in greater masses in different places. Strips of skin taken from the hands of the dead body of an individual who died of Addison's Disease (and whose case is published by M. Second-Ferréol), presented, under the microscope, the characters of the skin of the negro, showing a profusion of pigmentary granules.

Alongside the patches of a more or less decided brown color, Dr. Addison remarked, that others were visible which were not only lighter in shade, but were even of a dull white color, spots of true vitiligo, mingling with the brown patches, just as if the pigmentary matter had deposited itself in some places and had neglected others.

As the disease advances, the bronzed color becomes more decided, while, at the same time, the general symptoms assume increased intensity. Day by day, the debility increases; and the individual, having become wasted to the last degree, dies from exhaustion, unless he be abruptly carried off, like our patient, by an acute intercurrent affection.

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[1] HÉRARD:—*Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris:* 1867.

All these general symptoms resolve themselves into those of extreme anæmia. Professor Charles Robin on examining the blood of our patient (a small quantity having been taken by a cupping-glass) found, that it presented nothing abnormal, except a diminution in the red globules, such as is always observed in anæmic subjects.

In two patients, however, observed by Dr. Siredey, the red globules were as numerous as in health, and presented no modification either in form or volume. The number of the white globules was diminished. There was an absence of pigmentary matter. In relation to this latter point, Dr. Siredey justly remarks that it is one of curious inquiry.

Dr. Siredey points out that the two patients, who were absolutely cachectic did not present the vascular blowing sounds which belong to anæmia, a fact which led him to conclude that the bronzed disease, classed by some among the anæmic affections, must be a peculiar species.[1]

The same observer has likewise pointed out a fact which demonstrates depression of vital power—that fact being a decrease of temperature. In three patients whom he observed, the temperature, when fasting, was always below 37° C., the normal standard: in one subject, it was 35·7°, in another, 35·4°, and in a third 36°. These phenomena are interesting, they have not often been observed, and demand further investigation.

The urine of our patient, which was examined by M. Robin contained some pus-globules, but they were so few in number as to possess no pathological importance. In the urine of his patients, Dr. Siredey found traces of glucose, and cyanurine, but no appearance of albumen.

Dr. Jaccoud, in a work of remarkable merit, like every work of that distinguished physician, from an analysis of 127 cases of the bronzed disease, thus characterises it:—" An asthenia which goes on increasing up to death, a melanodermia presenting special characters, gastric disturbance, pains in the loins and abdomen—such are the four groups of symptoms which essentially constitute the symptomatology of Addison's Disease. The two first are constant, and may continue during the whole course of the disease: the others occur sufficiently often to be characteristic, and to be useful aids to diagnosis."[1] The gastric derangement consists in obstinate and sometimes quite intractable vomiting: this symptom was present in 74 of the 127 cases analysed. It was absent in my two patients, and in the three patients of Dr. Siredey. Along with the symptoms now enumerated, Dr. Jaccoud groups others which like them are dependent on perturbation of the nervous system, viz., headache, convulsions, vertigo, delirium, and coma. These symptoms are by no means of frequent occurrence.

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[1] Siredey:—*Bulletins et Mémoires de la Société Médicale des Hôpitaux*, p. 355: Paris: 1867.

Connected with animal life, Dr. Jaccoud observed palpitations and dyspnœa: in three cases, they were expressly pointed out, and in many others, no doubt, existed, but escaped notice. Dr. Jaccoud called attention to the fact, that while the asthenia was so serious as to lead to death, it was not accompanied, as might have been expected, by loss of flesh, albuminuria, leucocytosis, hemorrhages, vascular blowing sounds, and diarrhœa, as are met with in persons affected with cachectic asthenia. When there is loss of flesh, it is attributable to pulmonary phthisis: albuminuria is very unusual, and, as you know, was absent in our patients, as it was likewise in the patients of Dr. Siredey. Leucocytosis is exceedingly seldom met with, and is due to a complication and not to Addison's Disease. Hemorrhages were observed in only one patient; and he had disease of the liver. Dr. Siredey has not found vascular blowing sounds except in patients who had become anæmic from anterior or concomitant affections. Diarrhœa, which as you saw, terminated the life of one of our patients, is attributed, in the few cases in which it occurred, to complications independent of the bronzed disease.

The *prognosis* is shown to be of the most unfavorable possible character by the cases related by Addison and other physicians, by that of our patient, and also by two other cases which I have recently had to treat. When the disease, after pursuing a chronic course, becomes acute, death is the inevitable issue.

This is also what happens in leucæmia, as in other kinds of extreme anæmia of very long duration, and which neither originate in great loss of blood, bad or insufficient food, nor in poisoning by marsh miasmata. Persons affected by the bronzed disease have never been known to recover.

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[1] JACCOUD:—*Dictionnaire de Médecine et de Chirurgie Pratiques:* T. V., Article, BRONZÉE (Maladie).

At the *autopsy*, there are found, almost invariably, lesions of the supra-renal capsules; and, in most cases, tubercle, cancer, and fibrous products. It is a remarkable circumstance, that Addison, and those who after him reported cases of the disease which he was the first to describe, never met with the hemorrhages of the capsules, and consecutive capsular dilatation, as pointed out by Rayer in his memoir to which I have alluded. Sometimes, cancerous or tuberculous deposits were found both in the capsules and in other viscera, while at other times, the capsules only were affected. The kidneys were generally healthy, and almost free from any modification of structure.

In all cases of the bronzed disease, the supra-renal capsules are in a morbid condition; but the converse is not a correct statement, for there may be lesions of the supra-renal capsules without bronzed disease. Addison explicitly says so, quoting, with that good faith so conspicuous in his work, a case in which, on making an autopsy, he found small multiple cancers, and cancerous products in the supra-renal capsules of a patient who had never had in the least degree the special tint of the skin. Rayer also cites cases which are analogous in this sense, that the supra-renal capsules were found more or less completely destroyed in persons who had never presented symptoms characteristic of Addison's disease.

Here, then, Gentlemen, to the already extensive catalogue of chronic diseases, we have to add a new and entirely distinct species of anæmia. Need I say, how great is the service which Addison has rendered to our science and art by establishing this important distinction!

When I began my medical studies forty years ago, the subject of dropsy was an inextricable chaos. Fortunately for practice, this confusion has been unravelled by the beautiful researches of Professor Bouillaud, my colleague, and Dr. Bright. The researches of Virchow and Bennett into the subject of leucæmia, and of Addison into the bronzed disease, have thrown a new light upon the not less complex question of anæmia. Till then, the treatment of anæmical affections was deplorably empirical, it being impossible to distinguish cases in which we might usefully interfere from those in which no treatment could be of any use. In dealing with the former, we might make lucky hits; but in treating the latter, we incurred the risk of seriously complicating already serious cases by employing unsuitable measures.

Nevertheless, Gentlemen, the diagnosis of the disease now under our consideration is far from being devoid of obscurity. The special color of the skin, which constitutes its pathognomonic character does not generally make its appearance till an advanced stage of the disease. Its beginning is announced by a series of phenomena which belong to it in common with other forms of anæmia.

We must beware of mistaking for Addison's disease other cachexiæ in the course of which the skin assumes a dirty hue, considerably resembling that which is observed in the *bronzed disease.* Thus, in pregnant women, and in individuals with profuse and protracted suppurations, the skin acquires a dirty or brown appearance; but this coloration is not distributed in the same localities as in Addison's disease, nor is there ever that bluish color of the mucous membrane of which I spoke.

The remarks which I made on the prognosis render it unnecessary to enlarge on the *treatment.* All the means which have been tried have failed to prevent a fatal issue. Having no specific remedy, we are obliged to direct our measures against the symptoms of anæmia: ferruginous medicines, preparations of cinchona, and a tonic regimen, which you have seen me employ in the case of our patient, are indicated.

My friend, Dr. Duclos, lays down similar therapeutic rules.[1] My talented colleague of Tours is of opinion that the *bronzed disease* is dependent on a lesion of the supra-renal capsules; that the symptoms, the progressive debility so characteristic, and the fatal issue of Addison's disease, are consequences of a general poisoning of the economy by the pigmentary matter, which, in consequence of the perturbation of the function of the supra-renal capsules, is not destroyed as it is under normal conditions. Dr. Duclos founds his opinion, which he discusses with his usual ability, upon the attentive study of the case which he observed, and the cases recorded by authors: he also founds his opinion upon the results of the physiological experiments of Brown-Séquard and Vulpian—experiments which he looks upon as in accord with the information he had obtained from clinical observation.

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[1] Duclos:—*Bulletin Général de Thérapeutique,* for 1863.

I am well aware, and it is my duty to tell you, that this view of the disease has been met by many objections. Dr. Martineau in his inaugural thesis on Addison's disease,[1] while he regards it as a well defined morbid entity, disputes the part assigned to the supra-renal capsules. He rests his opinion on cases, derived from different sources, in which the disease had existed, while no trace of lesion in the supra-renal capsules had been found on examination after death.

Dr. Jaccoud has enunciated the following theory of the pathological chain of symptoms in Addison's disease. The rich supply of nerves which the supra-renal capsules possess, and the presence of ganglionic corpuscles in their medullary substance, justify our regarding them as a nervous apparatus dependent upon the abdominal sympathetic system. But the semilunar ganglia constitute the centre of innervation, and consequently of reflex action of the supra-renal plexus: all abnormal excitation then of the one will affect the other by reflex action producing the visceral disturbance which belongs to Addison's disease. On the other hand, the great sympathetic holds under its influence the vaso-motory system: this system presides over calorification and pigmentary secretion. Now, one cannot understand reflex disturbance of the vaso-motors on the one hand producing that reduction of temperature pointed out by some observers (and recently by Dr. Siredey), and on the other hand, that exaggerated secretion of pigment which is the cause of the bronzing. To explain the examples of incontestable lesion of the capsules without bronzing of the skin, and bronzing without lesion of the capsules, Dr. Jaccoud remarks, in relation to the first order of facts, that visceral disturbances are produced more easily and more quickly than the deposit of pigmentary matter, so that they may have been of sufficient intensity to cause death before it was possible for the bronzed color to have been produced: in respect of the second class of facts, he adds, that other alterations of the nervous plexuses might excite the abnormal secretion of pigment, and that thus we might have bronzing without lesion of the supra-renal capsules. Moreover, remarks Dr. Jaccoud, bronzing without progressive asthenia and without visceral disturbance is not Addison's disease.

Without criticism, I leave this ingenious pathogenesis in your hands.

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[1] Martineau (L.):—De la Maladie d'Addison: avec trois planches coloriées. Thèse de Paris, 23 Décembre, 1863.

Professor Sée regards Addison's disease as a cachexia, the seat of which is probably in the hematopoietic organs; and which is generally of a tuberculous or cancerous character. The nervous phenomena, and the very great prostration which accompany this cachexia, seem to be explained by the nervous texture of the supra-renal capsules. When there is a lesion of these organs, it is supposed that the alteration is nervous: when there is an absence of material lesion, we are obliged to fall back on neurosis of the capsular nerves. According to Professor Sée, it is most rational to suppose that there is disturbance of function in the vascular glands, particularly in the supra-renal capsules.[1]

My personal experience is too limited to allow me to come to an absolute opinion upon this interesting question; and I doubt whether others are really better prepared to speak in a fashion more categorical and positive. However, from what I have seen and read, I rather incline—as I have already said in this lecture—to the theory held by Dr. Duclos, and to believe that there is a relation between the lesions of the supra-renal capsules and the bronzed disease.

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[1] Sée (G.):—Du Sang, et des Anémies. Paris, 1866.

LECTURE XC.

LEUCOCYTHÆMIA.

A Disease characterised by great and progressive augmentation in the white globules, or globulines of the blood.—In Leucæmia, there is Enlargement of the Spleen, Lymphatic Glands, and Liver.—Etiology entirely unknown.—The only Essential Symptom of the Disease is the Presence in the blood of a great number of Leucocytes and Globulines.—Anæmia and Cachexia are consequences of Leucæmia.—Preparations of Cinchona, which have so manifest an action on Engorgements of the Spleen caused by Marsh Miasmata, have no effect on Engorgements of the Spleen in Leucæmia.

Gentlemen:—Chemistry and the microscope are often of very doubtful utility in their applications to pathology, and still more in their applications to therapeutics. Notwithstanding the progress which is being made day by day, notwithstanding the efforts made by eminent men who specially occupy themselves with these means of investigation, we too frequently discover their worthlessness at the bedside of the patient. Still, let me at once admit, that, under certain circumstances, they have rendered, do render, and will continue to render, signal service: in some cases, indeed, it is absolutely necessary to employ them as means of diagnosis. Was it not by these methods of investigation demonstrating the presence of sugar in the urine, that glucosuria became better known to our generation than it was in former times? And without employing them, when this disease presents itself to our notice, how should we be able to attain a precise knowledge of the case? Similar remarks are applicable to albuminuria: it is chemistry which enables us at any moment to seek for and find albumen.

As much may be said of the utility of the microscope. Docs it not give us, on very many occasions, information regarding the normal and pathological anatomy of the different tissues of the body? What services is it not destined to render to the study of pathological anatomy, which it has started on a career of true progress. Therefore, while we avoid falling into the excesses of those who believe that everything in pathology is to be achieved by the aid of chemistry and the microscope, who would allow them to dominate medicine, who through them would rationalise therapeutics, it is important that we study them, as means of clinical investigation, so as to be able to utilise them on fitting occasions.

It is to the microscope, and to the happy application made of it by Hughes Bennett and Virchow in the diagnosis of leucocythæmia, that we owe the power we now possess of distinguishing hypertrophy of the spleen, which is characteristic of this malady, from hypertrophy of the spleen symptomatic of paludal poisoning, of the paludal diathesis. We required to have recourse to the microscope to establish a precise diagnosis in the case of a man who occupied bed 9, Saint-Agnes's ward. I hesitated some time before I formed a settled opinion as to the nature of his case. Some of you thought, with one of my most skilful colleagues, that, possibly, this man had renal disease; others, looking to the fact that the patient came from a district where intermittent fever was endemic, believed (although the patient affirmed that he had never had intermittent fever) that there was hypertrophy of the spleen consecutive on paludal poisoning; but the microscope relieved us from the uncertainties of our diagnosis, by demonstrating the existence of that alteration of the blood which is characteristic of leucocythæmia.

When healthy blood is examined by the aid of the microscope, we observe (besides the red globules, which are seen lying one above another like a pile of five franc pieces), other globules which are white, larger in diameter, and much less numerous than the red. We also find isolated nuclei, in a proportion which may be described as insignificant.

In certain physiological conditions, such as during digestion, menstruation, and pregnancy, and also, in certain pathological states, as in inflammatory diseases, in typhoid fever, puerperal fever, cancer, and phthisis, when these maladies are far advanced, there is an increase in the number of the white globules of the blood; but this numerical increase, essentially temporary in the physiological state, essentially accidental in pregnancy, being in all these instances subordinate to causes which are not persistent, does not constitute leucocythæmia, any more than diabetes is constituted by the accidental presence of sugar in the arteries and veins, in the renal arteries, and sometimes even in the urine, during digestion.

This increase of white globules in the diseases I have mentioned, no more constitutes leucocythæmia, than the presence of albumen in the urine in the first stage of cholera, in convulsions, and in inflammatory sore throat, constitutes Bright's disease.

To constitute leucocythæmia, that is to say, the special disease, the dyscrasia, which, from its very beginning makes incessant progress leading to inevitable death, it is necessary that the excess in the proportion of white globules be greater than in any of the circumstances I have mentioned: the maximum proportion fixed by authors who have treated this subject is one to twenty.

According to Moleschott, in the normal state of the economy, the proportion of white to red globules is 1 in 346. In leucocythæmia, the proportion is at least 1 in 20: between this minimum and the report of 1 to 1 noted by Dr. E. Vidal, my colleague in the hospitals,[1] cases have been met with in which the proportion was 1 to 19, 1 to 12, 1 to 7, and 2 to 3. But according to Virchow, leucocythæmia does not exist, merely because there is a certain increase of the white globules: he holds, that there must also be a simultaneous diminution of the red globules, a substitution of the former for the latter—this substitution being often so great that the blood assumes a more or less white color, as if (says the German author) there was true albinism.[2]

When we wish to examine the blood of an individual whom we suspect to be stricken by leucocythæmia—and I repeat, it is only by a microscopical examination of the blood that we can arrive at a precise diagnosis—we prick with a needle the end of one of the patient's fingers. The blood, on exuding, has a troubled appearance and a yellowish red color: on coagulating, it assumes a deeper brown hue. In a case published by Vogel, blood drawn from a vein was in two separate portions. The first was defibrinated: after four hours, a whitish cream was seen floating on the surface, and after twenty-four hours, the defibrinated portion of blood divided into two layers, the upper being of milky whiteness resembling pus, and the lower being of a reddish brown colour. The second portion coagulated like healthy blood: the clot was covered by a whitish granular layer formed by the aggregation of the white globules: the serum was abundant, clear, and limpid. This experiment of Vogel recalls to our recollection, the process of separation pointed out in 1844 by Donné.[1] This process consists in defibrinating the blood, and allowing it to settle till the white globules have separated: the white globules, being less dense than the red, float, while the red are precipitated. By proceeding in this way, results are obtained, which though not absolute, at least admit of easy comparison. Dr. E. Vidal has turned to account this method, which is applicable to a very small quantity of blood, for estimating the relative proportions of white globules which exist in a case of leucocythæmia.[2]

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[1] VIDAL (E.):—De la Leucocythémie Splénique. [*Gazette Hebdom. de Médecine*, 1856.]

[2] VIRCHOW (R.):—*Gesammelte Abhandlungen zur Wissenschaftl. Medezin*: *Frankfurt*, 1855.

The blood, having been first defibrinated, was poured into a graduated tube. Forty-eight hours elapsed before complete separation had taken place. The mass was then seen to be divided into three well marked layers differing in thickness.

First, there was an upper layer formed by the serum, which was limpid, lemon-coloured, and normal in appearance.

There was then formed a middle layer of grizzly-yellow, slightly inclined to green, of a color analogous to that of pus, constituted by the aggregation of white globules.

Lastly, the under layer was composed of red globules of the color of wine-lees, marbled towards its upper part, and exhibiting some whitish particles adherent to the sides of the glass.

In a first examination, the second layer (white globules) was in relation to the third (red globules) in the proportion of one to 2·14. Six months later, a renewed examination was made, when it was found that the layer of white was greater than the layer of red globules, their relative height being in the proportion of 1·25 to 1.

On counting the globules in the field of the microscope, it was found that there was a nearly equal number of white and red, the latter, however, being rather more numerous. To appreciate properly the apparent difference between the results of the process of separation and the numerical estimate, we must bear in mind that the leucocytes are more voluminous than the red globules above which they float; they are less piled up on one another, and there is interposed between them a certain quantity of serum.

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[1] Donné:—Cours de Microscopie.

[2] Vidal:—*Bulletins de la Société Anatomique:* 1857.

Considering that this alteration of the blood, this substitution of leucocytes for red globules, is coincident with the alteration of the solids which we are now about to study, and granting that the disease began with special lesions of the spleen and other vascular glands, lesions which show themselves by the organs becoming hypertrophied before the blood has undergone the characteristic change, Bennett and Virchow have enunciated an essentially different theory of the nature of leucocythæmia.

According to Virchow, the spleen and lymphatic glands are charged with destroying, in a certain manner, the red globules. The more these organs are hypertrophied, the greater, he says, is their activity; and consequently, the number of white globules will be great, and the number of red globules will be diminished.[1]

Bennett, holds that the spleen and lymphatic glands are charged with the formation of the white globules; but he does not admit that these organs destroy red globules. He believes that red globules are really white globules modified and colored in other parts of the circulatory system. The difference between the two theories is, therefore, at once apparent. According to Bennett, there is increased functional activity of the spleen resulting from its hypertrophy, leading to the formation of a number of white globules, which ultimately circulate in so great a quantity that the transformation of all of them into red globules is impossible. According to this theory, there is no substitution of white for red globules, but only an increase in the proportion of the white globules.

Dr. E. Vidal and Professor Magnus Huss discuss both of these theories and consider neither satisfactory, because, on the one hand, they are based on physiological views of a merely theoretical character being demonstrated to be truths, and on the other, because, if leucocythæmia be exclusively dependent on hypertrophy of the spleen, why is it not associated with the hypertrophies consequent upon intermittent fevers, and in some of which the spleen acquires a volume at least as great as in any case of leucocythæmia? You may recollect the young woman who had contracted a quartan fever at Guadaloupe, and came into our wards with a greatly enlarged spleen. Her blood did not contain an excess of leucocytes. It is evident, then, that there must exist, besides the hypertrophy, a hitherto undiscovered special structural and functional lesion of the spleen.

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[1] VIRCHOW:—"La Pathologie Cellulaire basée sur l'Etude Physiologique et Pathologique des tissus;" traduit de l'allemaud par Paul Picard: Paris, 1861.

The same objection may be made to the lymphatic leucocythæmia of Virchow. Though this kind of leucocythæmia is characterised, according to the Berlin professor, by hypertrophy of the lymphatic glands, the spleen preserving its normal size and structure, it differs from splenic leucocythæmia, not only in the white globules predominating in the blood, but also in the predominance of globulines identical with those of lymph. Well then, if in this kind of leucocythæmia, hypertrophy of the lymphatic glands were the sole cause of the disease, how are we to explain the fact, that it does not always occur in the many cases in which we find glandular engorgement and hypertrophy without leucocythæmia? We had lately an example of this in a tuberculous patient who died in our wards with great enlargement of the cervical glands. His blood, examined by the aid of the microscope, presented none of the morbid alterations which belong to the disease we are now considering. Moreover, in an early lecture, I will lay before you several cases of generalised glandular hypertrophy, in which neither an excess of white globules nor of globulines had ever been detected by microscopic observation.

To sum up:—we may say with Vidal and Magnus Huss that, while we admit leucocythæmia to be a malady *sui generis*, we possess no satisfactory data whereby to determine its essential nature; and that the close relation which may exist between morbid change of the spleen or glands and morbid change of the blood will remain unknown, so long as the formation of the blood, and the functions of the spleen and glands without excretory ducts (such as the thyroid and thymus) remain secrets.

In pointing out the alterations of the blood characteristic of leucocythæmia, I have noticed the most important part of the anatomical history of this cachexia. I have, however, still to speak of the state in which the blood is found on opening the dead body. Its color varies from brick-red to a deep brown or chocolate hue. Sometimes, it forms clots which do not adhere to the walls of the vessels which they fill to distension; but there is never any alteration in the walls of the vessels. The clots are mingled with yellowish or greyish coagula, which at a first glance may be mistaken for concrete pus. Sometimes, we find the blood fluid, pale, and reddish yellow, having the appearance of the muddy blood of the spleen, and containing a large quantity of white globules.

The most remarkable organic lesions are found in the spleen, liver, and lymphatic glands.

The increase in the volume of the spleen, which in the great majority of cases of leucocythæmia arrests the attention of the physician in the living patient, exists nearly always, or, I may say, always, in the dead body.

The weight of this organ reaches as much as six [French] pounds; and its dimensions, which generally vary between 30 and 32 centimeters in length, between 16 and 18 in breadth, have been as much as 41 by 20 centimeters, with a thickness of 7 centimeters. Its form is an enlargement of the natural form: in different cases, there is a great diversity in the appearance and consistence of its parenchyma. In the majority of cases, the tissue is hard, frangible, of a uniform deep-brown or reddish brown color, and exhibiting, on section, a lustrous aspect: in some cases, the color tends towards yellow, presenting red and yellow layers, which give it a marbled appearance. In five autopsies, included in the history of the cases of leucocythæmia which form the basis of Dr. Vidal's excellent monograph, the spleen contained one or more deposits of whitish or yellowish white matter resembling the fibrinous deposits sometimes met with in its interior in subjects having disease of the heart.[1] In two cases, it was riddled with small whitish points. Its capsule was thickened, opaque throughout, and adherent by plastic exudation to the diaphragm and peritoneum.

Examined under the microscope, its tissue presents important changes; viz. increase in the number and volume of the normal elements, and the substance between the cells of the pulp is more abundant and more condensed than in the healthy state. This modification of texture has been described by Virchow under the name of *hyperplasia with induration.* Vidal and Luys have observed great hypertrophy of the Malpighian tufts. These tufts filled with cells having several nuclei, and with free nuclei, are trippled or quadrupled in size, and assume a whitish appearance, marbling in a certain manner the reddish brown of the parenchyma of the organ.

The liver, without being altered in structure, is increased in volume. In some cases, it attains three times its normal bulk, and weighs from four to six kilogrammes.

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[1] VIDAL (E.):—De la Leucocythémie Splénique. [*Gazette Hebdom. de Médecine*, 1856.]

The lymphatic glands, which are often hypertrophied, never present, even in lymphatic leucocythæmia, more than simple augmentation of their normal elements.

Gentlemen, let me call your attention to an anatomical fact recorded by Dr. Lancereaux. While it tends to confirm the remarks of Magnus Huss, E. Vidal, and Virchow, it also shows us how numerous the white globules may be in the capillaries, particularly in those of the brain. The case occurred in M. Marotte's service at the Hôpital de la Pitié. The patient was thirty-two years of age, and had presented the symptoms of leucocythæmia. The liver and spleen were both much enlarged; and an examination of the blood showed that the white were very numerous, and also more numerous than the red globules. At the autopsy, it was found that the spleen extended from the hypochondrium to the symphysis of the pubes. It had a pretty uniform brownish color, which became yellow or bright red on exposure to the air: when cut, there flowed from it a very thick chocolate colored fluid, composed to a great extent of white globules. A tear of the organ presented a granulated appearance, and enabled one to see the hypertrophied Malpighian bodies, even by the naked eye. The cerebral sinuses and their affluent veins were filled with brownish clots. The surface of the brain presented a fine injection of the veins of the pia mater, a fact to which I wish to direct your special attention. It had the appearance of a mercurial, or, still more, of a purulent injection. The white matter which constituted this capillary injection was entirely composed of white globules. I am not aware, Gentlemen, that a similar injection of the capillaries of the brain had been observed in any previous case. The same kind of injection may probably be met with in other organs rich in capillary vessels, such as the lungs and glands.

It has been correctly remarked by Professor Sée, that enlargement of the spleen and lymphatic glands is not sufficient to produce leucocythæmia: he holds, that to produce leucocythæmia there must be *hyperplasia* of the proper tissue of the organ, that is to say, an augmentation of its active part. He says:—"If the spleen or lymphatic glands undergo a morbid change, without there being hyperplasia, no increase of white globules takes place: we meet with fever patients having enormous enlargement of the spleen, without any trace of leucocythæmia: these engorgements are sanguineous infarctus, or very formidable lesions of the tissue of the spleen." The same able physician also observes, that "leucocythæmia always supposes the formation of new glandular tissue, or of new elements in the normal glands: in the adult, leucocythæmia is *almost* always of splenic origin, while in the child, on the contrary, it is of glandular origin."[1]

Professor Sée adds that, when there is no primitive hyperplasia of the lymphatic glands, new lymphatic glands make their appearance, being formed everywhere in the pleura, liver, kidneys, and intestines. There is, so to speak, proliferation of the adenoid tissue, the lymphatic elements being increased in number and volume: this is the conclusion derived from the observations of Friedreich, Leudet, Botcher, and Billroth.

Such are the principal anatomical characters of a disease of which we received the first accounts almost simultaneously from Germany and England, where they were published in 1845, at an interval of only a few days, by Virchow[2] of Berlin and Bennett of Edinburgh.[3] In France, however, so far back as 1836, the malady was observed by our able colleague, Dr. Barth, as is mentioned by Dr. Vidal in his work, in the chapter devoted to the history of the subject. In 1852, Dr. Leudet published a case of leucocythæmia, in which the diagnosis was made after death;[4] and in the following year, Dr. Charcot placed another case on record.[5] Dr. Vidal's monograph, which I have frequently referred to, is based on thirty-two cases. To these, other cases might be added—particularly, the one related in the work of Professor Magnus Huss of Stockholm, the case now in our clinical wards, and the case of a child fifteen months old, admitted to our nursery ward in 1862, of which I am about to speak.

Dr. Vidal has remarked that leucocythæmia must be very rare in early infancy, as he had not found one case of its occurrence at that period of life, although he had made himself acquainted with most of the works which had appeared on the disease up to 1856. Of the thirty-two patients whose cases he analyses, the youngest was 13½ years of age. This is the proper place to narrate the case of the child of fifteen months, to which I alluded a minute ago. In that case, there was great hypertrophy of the spleen; and microscopic examination showed us numerous large white globules in the blood. To give to this case its full value, let me add, that Dr. Vidal was so good as to examine the blood of our little patient; and, on two occasions, he found that it presented all the characteristics of splenic leucocythæmia. Here, are the details of this case.

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[1] Sée (G.):—Du Sang et des Anémies: p. 280. Paris: 1866.

[2] Virchow:—*Froriep's Notizen:* No. 780.

[3] Bennett (John Hughes): *Edinburgh Medical and Surgical Journal,* for October, 1845.

[4] Leudet:—*Bulletins de la Société Anatomique:* 1852.

[5] Charcot:—*Comptes Rendus des séances de la Société de Biologie, p.* 44, 1re *Série, T. V,* for the year 1853.

A child, aged 15 months, suckled by its mother, was admitted, on the 16th February 1862, to Saint-Bernard's ward. It seemed to have been ill for a long time: the face was somewhat puffy: the mother stated, that for three weeks the child had had diarrhœa and vomiting: she likewise mentioned that it had had, for eight or ten days, every afternoon, an attack of fever with slight shivering. There were no signs of pulmonary phthisis nor of rickets. The extremities were thin and wasted: and the general wasting was rendered more obvious by contrast with the enormous size of the abdomen. By palpation, a very considerable enlargement of the liver was detected; and in the left hypochondrium, I found great tumefaction of the spleen, that organ descending obliquely as low down as the spine of the ilium, and its inner margin reaching nearly to the umbilicus. There was a small amount of ascites. As I have already stated, the little patient had had diarrhœa for some days: the mother had remarked that occasionally there was blood in the stools, and this I also noticed several times. Here, let me remark, that this child was born at the Hospice des Cliniques, had never been out of Paris, and, in all probability, had never been subjected to the causes which engender paludal fever. Microscopic examination of the blood revealed the presence of a great number of white globules. There was no room for doubt, therefore, as to the nature of the disease: it was certainly a case of leucocythæmia. During the month, the child remained in our wards, the diarrhœa and vomiting were kept in check by preparations of chalk and bismuth. The ammonio-citrate of iron seemed to impart a certain amount of color and firmness to the flesh. However, from time to time, the sanguinolent diarrhœa reappeared. Crude quinine, in doses of 15 centigrammes, did not always succeed in cutting short the paroxysms of fever which returned nearly every day, sometimes with, and sometimes without, shivering. There was no diminution in the volume of the spleen;

and when the mother wished, contrary to my counsels, to leave the hospital, it still extended as low down as the anterior iliac spine; and, on palpation, presented as great a degree of hardness as before.[1]

Gentlemen, I have already mentioned incidentally the slight influence which quinine has upon engorgement of the spleen in leucocythæmia. Let me now remind you of the great rapidity with which these engorgements disappear if produced by paludal poisoning, when treated by cinchona in large doses according to Sydenham's method.

At the same time that the child of whom I have been speaking was in our wards, you may remember to have seen, in bed 13 Saint-Bernard's ward, a young woman of 18, who came in with quartan fever and great engorgement of the spleen. Every time that I gave her cinchona, you saw that intense fever was set up for some hours, while at the same time, we could perceive that there was enlargement of the spleen. But from the following day, the fever entirely ceased, and the progressive diminution of the organ was clearly demonstrated by palpation and percussion. This diminution continued for six, seven, eight, or ten days, that is to say, during the whole of the apyrexial period; but as soon as the fever returned, the spleen was observed to swell anew. This almost experimental observation, which I repeated several times during the patient's residence in hospital, affords additional confirmation of two already known facts, viz. that cinchona has a special action on engorgements of the spleen of paludal origin, and that this result is not obtained without a temporary increase of the fever, accompanied, probably, by hyperæmia of the spleen.

No definite information has as yet been obtained as to the causes which give rise to leucocythæmia. In Dr. Vidal's thirty-two cases, it occurred twice as frequently in men as in women. The age of the patients has varied between fifteen months and sixty-nine years; but the affection is most common among adults. The ill fed and badly lodged, the poor, those addicted to excess in alcoholic drinks, and persons placed under unfavorable hygienical conditions, are those who have chiefly paid tribute to this sad disease.

On inquiring into the antecedents of the patients, we find that four women dated the beginning of their malady from the period of their last pregnancy. Others had previously complained of rheumatism. Some patients had had intermittent fever; but in these cases, no necessary relation could be established between paludal poisoning and the leucocythæmia. For even supposing, with Dr. Magnus Huss, that the splenic engorgement of paludal poisoning was in these exceptional cases an organic cause determining leucocythæmia, we must admit the small importance of such an etiology, when we observe every day splenic engorgements of various origin, which are not accompanied by any augmentation in the number of the white globules of the blood.

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[1] Vidal (E.):—De la Leucocythémie Splénique. [*Gazette Hebdomadaire de Médecine*, 1856.]

However, as a symptom, engorgement of the spleen has much importance: indeed, the circumstance upon which our patient laid most stress was the greatly swollen state of that organ. It is the phenomenon which, when present, enables the physician to make a decisive diagnosis between splenic and lymphatic leucocythæmia, because it is absent in the latter. Enlargement of the spleen is often also the fact to which patients attach most importance: this enlargement may, as I have said, attain great proportions. The abdomen bulges out in the hypochondrium and left side, in consequence of the enlarged spleen invading a great part of the abdominal cavity. The skin in that region is ridged by distended veins. The limits of the organ are easily ascertained by palpation: in our case, we could ascertain its limits by inspection. The tumour, in its upper portion, is fixed, and moves very slightly when the patient moves: it descends somewhat when the patient stands. Palpation and percussion occasion more or less pain; and sometimes pain spontaneously supervenes of so acute a character as to necessitate medical intervention. When patients are simply standing, they only experience a feeling of weight which is increased by walking or by the performance of any work. This feeling, being increased by the pressure of the clothes, obliges patients to slacken the waist-band.

Enlargement of the liver often coexists with hypertrophy of the spleen; but it is chiefly in the second period of the disease that enlargement of the liver is met with. It is accompanied by pain. Hypertrophy of the spleen and liver may give rise to abdominal effusion, and anasarca; but the latter serous effusion may occur at an advanced period of the disease under the influence of the cachexia.

Hypertrophy of the spleen or lymphatic glands, and the pathognomonic alteration of the blood—particularly the latter—are the symptoms which really belong to leucocythæmia: the other symptoms are not peculiar to it, and occur in all cachectic diseases.

In the beginning of the disease, there is debility which sometimes makes rapid progress. There is, at the same time, loss of flesh, paleness of the skin, and all the symptoms of anæmia, such as palpitation, buzzing in the ears, dimness of vision, headache, and sometimes a tendency to faint. Some patients complain of neuralgic pains. The temper becomes irritable, melancholy, and morose. In the latter days of the disease, a tranquil delirium supervenes, which continues till the death of the patient.

Digestion is generally well performed, even in the last stage of the disease; and though at the very end, diarrhœa is the most constant symptom, the stools generally remain regular, except in some individuals in whom there is an alternation of confinement and looseness of the bowels. However, in the case of a Spanish merchant, who consulted me in 1861, the malady began with disturbance of digestion: two or three hours after eating, he experienced acute pain in the stomach. He drank, without benefit, mineral waters of the most varied kinds, for the cure of this gastralgia: in vain, he changed the hours of his meals and the nature of his food—the digestion remained unimproved. It was not till three years had elapsed, that glandular enlargement made its appearance: treatment by preparations of iodine proved of no avail. The abdomen became distended with gas; and cachexia began to show itself. It was in these circumstances that the patient applied to me. I found that he had great enlargement of the spleen and liver, as well as of the lymphatic glands of the neck, axilla, and groin. On percussing the abdomen, slight ascites was discovered: the countenance was pale, thirst was urgent, and urine scanty. The pulse was quick, particularly at night. My accomplished colleague, M. Robin, on examining the blood at my request, found that the white globules were in the proportion of from 20 to 25 in 300, in place of 1 in about 300, the usual proportion in normal blood. The result of this examination, therefore, fully confirmed my diagnosis.

The embarrassed respiration, which is observed in the majority of patients from the beginning of the disease, and which is increased in some persons after dinner, by walking, or by movements of the body, is connected with anæmia, and is also probably dependent on the mechanical obstacle to the free play of the respiratory apparatus caused by the enlarged spleen, which pushes up the diaphragm into the cavity of the chest.

This dyspnœa, which increases as the malady progresses, may, in the last stage, become orthopnœa, although no pulmonary lesion can be found at the autopsy. It is sometimes accompanied by an occasional short cough, which is generally dry, though followed in some cases by slight mucous expectoration.

The pulse is weak and compressible; and does not become rapid till hectic fever is lighted up. In the cases in which this fever showed itself at an early period of the disease, it was very different from the paroxysms of intermittent fever. The febrile movement, however, often commenced with rigors, and was followed by profuse sweating, particularly at night, obliging the patient to change his linen: the attacks recurred very irregularly, and were very transitory: they generally came on towards evening, like paroxysms of hectic fever, and unlike paroxysms of paludal fever, which generally declare themselves in the morning or at noon.

I have mentioned anasarca, and have to a great extent attributed its occurrence to the cachectic state. Serous effusions into the cavities of the abdomen and pleura, œdema of the cellular tissue and lungs are generally observed in the last stage of leucocythæmia; and, in some cases, œdema has been seen to appear and disappear at different times.

It is usual to observe—as was observed in our patient—a tendency to hemorrhages. The most common are nasal hemorrhages, after which come intestinal, inguinal, and subcutaneous hemorrhages: in twenty cases, there were two in which metrorrhagia occurred.

Virchow connects this tendency to hemorrhage with the affection of the spleen. Here, again, apply the remarks I made on the supposed relations between the alterations of the spleen and blood; for, as was observed long ago, great enlargement of the spleen following paludal fevers usually gives rise to hemorrhages. I may also refer to structural change of the liver, it being well established, particularly since so much light was thrown on this subject by Dr. Monneret, that there is a tendency to hemorrhages when the liver is diseased. But, asks Professor Magnus Huss, may not the hemorrhages in leucocythæmia be more dependent on the excess of white globules? Being of larger diameter than the red globules, and having a tendency to become agglutinated, they will form clots obstructing the passage of the blood through the capillaries, and rupturing them: the result would be hemorrhage, more or less considerable in amount, according to the extent of the rupture of the vessels. I confess that this mechanical explanation does not much commend itself to me.

The urine is normal in the first stage of the disease; but towards the end, it contains ammonia and the urates in increased proportions.

In some cases, under the designation of complications, there have been noted concomitant affections of the lungs, such as tubercular disease, pleuritic effusion, sanguineous congestion, and œdema. In three of the cases detailed by Dr. Vidal, jaundice occurred during the course of the leucocythæmia: in another case, there was cirrhosis of the liver: and in three other cases, the leucocythæmia was complicated with Bright's disease.

Furuncular eruptions, with sloughing over the sacrum, and pemphigus (as in a case of Virchow and in another of Magnus Huss), have been observed to supervene during the last stage of leucocythæmia.

It is rather difficult to state with precision the duration of leucocythæmia, as we never know the exact time at which it began; but, speaking approximatively, we may say, that in the cases related by authors, the disease lasted from three months to five years, the average duration being from thirteen to fourteen months. Its progress, therefore, is essentially chronic: its termination is death. On this point, all observers are agreed: in all the cases which have been reported, the account states, either that the patient had died, had not been cured, or remained in an almost hopeless state.

Different methods of treatment have been employed; but they have all proved not only ineffectual as means of cure, but even of no avail in temporarily arresting progress. Nevertheless, I am inclined to think that some relief will result from treatment directed to the anæmic condition; it may not suffice to cure the disease, but it may retard its progress, if it be allowable to draw a conclusion from what we saw in our patient. The preparations of iron have proved very useful; and preparations of cinchona have been of undoubted benefit in arresting the hemorrhages. For some time after coming into hospital, our patient had every day rather alarming attacks of epistaxis: on having recourse to the powder of cinchona, the hemorrhages did not occur for some time. Every day, he took, in a little coffee, two grammes of the powder of yellow cinchona; for three weeks, he had no bleeding from the nose. But it recurred; and always yielded anew to the influence of a large dose of the same medicament. Notwithstanding the reality of this amendment, it was too slight to justify a more favorable prognosis.

To give you an idea of the medication which I recommend, let me quote the substance of the written advice which I gave to the Spanish merchant of whom I spoke a little time ago. I prescribed for him the waters of Pougues, recommending him to begin at once, if possible, to take them: I also recommended preparations of iron, and preparations of iodine, in the first instance alternately, and then combined—the successive use of saline, sulphurous, and ferruginous baths—powder of cinchona—wine of cinchona—bitters, such as quassia and nux vomica—and finally, a varied diet. The understanding was that the patient's Spanish physicians should treat him in accordance with this general programme, modifying details as circumstances changed.

LECTURE XCI.

ADENIA.

An Affection characterised by Progressive Hypertrophy of the superficial and deep Lymphatic Glands.—Hypergenesis of Glandular Cellules.—Never any Inflammation of the Glands.—Sometimes concomitant Hypertrophy of the Spleen, Liver, and Intestinal Glands.—The Disease has Three Periods: viz. the Latent, the period of Progress and Generalization, and the Cachectic Period.—In the first period, there is no general disturbance of the system: in the second and third periods, there is Anæmia without Leucocythæmia.—Œdema of the limbs, Ascites, and sometimes Anasarca.—Cough.—Dyspnœa.—Suffocative Attacks from compression of the Bronchiæ.—Duration of the Disease is from eighteen months to two years.—The Termination is almost always fatal, either by an attack of suffocation, or by the cachectic state.

Gentlemen:—From the earliest times, the attention of physicians has been directed to *engorgements*, or, as they were formerly called, *obstructions* of the viscera. In treating of the cachexiæ, the older writers attached great importance to obstructions of the spleen and liver.

Since the publication of the works of Sydenham, Morton, and Torti on paludal poisoning, there has always existed an anxious desire to ascertain its relation to enlargement of these organs. The anæmia and cachexia which accompany these visceral engorgements had led to the belief that there was an essential alteration in the constitution of the blood. Morbid changes in the constituents of the blood had been suspected, but not proved. You recollect with how much interest the works of Bennett and Virchow on a new disease were received—the disease to which they gave the name of leucæmia, and considered as the consequence of hypertrophy of the spleen, liver, or lymphatic glands.

At first, great enthusiasm was created by the discovery of the two foreign savants; and every body endeavoured to adduce confirmatory cases. It was believed that there was a leucocythæmia connected with the spleen, and a leucocythæmia connected with the lymphatic glands. Numerous cases, however, have proved that engorgement of the liver, spleen, and lymphatic glands may exist without there being any modification of the number of the white globules and globulines; and, moreover, the important researches of Professor Charles Robin have shown, that white globules and globulines may be in excess in the blood although neither visceral nor glandular hypertrophy exist. Moreover, hypergenesis of leucocytes may be met with in morbid states of the economy quite unconnected with any visceral engorgement, and even in physiological conditions.[1]

Gentlemen, it has been long known that hypergenesis of leucocytes is not a necessary consequence of hypertrophy of the spleen and liver. Clinical observation was not long in establishing that hypertrophy of the lymphatic glands might also exist without any increase in the number of globulines.

The subject to which I wish to direct your attention to-day is GENERALISED HYPERTROPHY OF THE LYMPHATIC GLANDS. Since my attention was first directed to this strange affection, I have been struck with never seeing suppuration of those enormous lymphatic tumors which for a time give rise to no other inconveniences than those arising from the effects of their pressing on organs.

I have also ascertained the incurable character of these tumors, which though they never became the seat of inflammation, possess insurmountable tenacity, constantly tend to increase in size, and after a period of very indefinite duration, end by producing a most injurious influence on the constitution, sometimes causing death by compressing organs essential to life.

These facts have convinced me that we ought to regard the formation of these glandular tumors as a specific form of disease. Wishing to attach a special name to a special disease, I called it ADENIA. This name, no doubt, might be applied to a great many glandular affections, such as cervical and mesenteric tuberculisation. Nevertheless, I persist in giving the disease this name, that it may henceforth be an established fact that a new morbid species has been found in the extensive family of glandular diseases.

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[1] *Journal de Physiologie de* BROWN-SEQUARD, p. 51. Paris: 1859.

But, Gentlemen, let me first of all beg you to understand, that I have made no discovery—that before I described the disease, it had been well described by others—in England, by Hodgkin;[1] in France, by Dr. Bonfils, one of my pupils;[2] and in Switzerland, by Dr. Cossy.[3] I lay claim to no other merit than that of having brought together a large number of cases collected from my own practice and from that of my professional brethren, associating them together by one common description, including them in one special name, and endeavouring to disseminate a correct knowledge of them.

My friend and colleague, M. Nélaton, has frequently addressed his pupils on the disease now under our consideration: and, like me, he has been disposed to regard it as a glandular affection quite special in its character. My colleague Professor Laugier (in whose hospital wards I saw the case reported by Dr. Bonfils) was equally struck with the peculiar characters of the disease. M. Leudet, the honorable and accomplished director of the Rouen school of medicine (with whom I have often discussed the subject), has remarked, with his usual sagacity, that adenia is a disease altogether special.

In 1861, Dr. Cossy published a memoir, in which he recorded three cases of simple general hypertrophy of the glands, accompanied by leucæmia. Finally, I am indebted to Drs. Potain and Laboulbène, my colleagues in the Parisian hospitals, for having been so exceedingly obliging as to communicate to me two cases, one of which occurred at the Hospice des Ménages and the other at the Hôpital Sainte-Marguerite.

All these cases greatly resemble one another; and, from their attentive study, it appears, that adenia consists in simple hypertrophy of the superficial and deep-seated lymphatic glands, and in the formation, in different organs, of lymphatic products analogous to those met with in leucocythæmia, but—and the fact is both essential and characteristic—unaccompanied by any augmentation in the white globules of the blood.

The hypertrophy of the glands is sometimes accompanied by simple hypertrophy of the liver and spleen. Moreover, as I have just told you, and as was very apparent in Dr. Potain's case, there is sometimes hyperplasia of the aggregate and solitary intestinal glands.

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[1] Hodgkin:—On some Morbid Appearances of the Absorbent Glands and Spleen. [*Medico-Chirurgical Transactions* for 1832: vol. xvii, p. 168.]

[2] Bonfils:—Réflexions sur un cas d'Hypertrophie Ganglionnaire Générale. [*Société Médicale d'Observation de Paris:* 1856.]

[3] Cossy:—Mémoire pour servir à l'Histoire de l'Hypertrophie Simple plus ou moins Généralisée des Ganglions Lymphatiques, sans Leucémie. [*Echo Médical*, T. V. Neuchâtel: 1861.]

In all the cases, excepting perhaps in one of those communicated to me by Dr. Leudet, there was no increase in leucocytes or globulines.

Adenia, therefore, is distinguished from other glandular affections, and particularly from leucocythæmia, by there being no appreciable alteration in the blood, excepting that which belongs to anæmia, and in the anæmia not showing itself till the second stage of the disease.

The patients generally apply for medical advice during the early months of the malady. They complain of numerous tumors on the surface of the body, and, sometimes, of slight dyspnœa. They state that they are in good health in other respects: there is no loss of appetite, no serious disturbance of the principal functions, and during the first five or six months of the disease, nutrition is not morbidly influenced in any appreciable degree.

Hypertrophy of the glands usually begins in the submaxillary region: soon afterwards, the patients are alarmed by observing the formation of tumors in the sides of the neck, in the axillæ, and in the groins. Less frequently, there is hypertrophy of the epitrochlean and popliteal glands. The enlargement of the submaxillary and cervical glands soon gives a singular appearance to the face: the head, which appears relatively small, rests on a glandular mass which the patients try to hide by adopting toilette artifices. The tumors in the neck are not associated with any change in the colour of the skin. They have not contracted adhesions with neighbouring parts, and it frequently happens that the individual hypertrophied glands remain quite free from each other. They are movable: they can be touched, squeezed, and even kneaded without causing pain. Those situate in the submaxillary region, joining with those of the opposite side and with the cervical lateral tumors, sometimes permanently assume the form of a band round the neck. The latter are not exclusively superficial. In one case, I have seen the lateral tumors on the sides of the larynx and trachea, and extending probably to the bronchi. They sometimes extend below the clavicle, and become continuous with axillary tumors. The axillary tumors are generally very large, being often the size of the egg of a hen or turkey. Drs.

Escalonne and Leblanc had a patient near Fontainbleau, in whom they were as large as mammæ, to which indeed they bore a strong resemblance, both in respect of the colour of the skin and the network of veins on their surface. Tumors so considerable in volume not only interfere with the movements of the arms, which they keep far apart from the trunk, but they likewise present an obstacle to the venous circulation, and not unfrequently also produce œdema of the hands and forearms. Sometimes, the subpectoral glands are enlarged.

The inguinal glands also become very large: consequences follow similar to those produced by the greatly enlarged axillary glands—that is to say, a difficulty in moving the arm, and an impediment to the free return of the venous blood. The feet and legs are nearly always œdematous. The inguinal tumors sometimes occupy the whole extent of the triangle of Scarpa; and often, the hand, when applied below Poupart's ligament, can feel similar tumors in the iliac fossæ. By introducing the finger into the vagina and rectum, similar tumors can be detected in the pelvis. In thin persons, by means of abdominal palpation, enlarged glands may be felt at the sacro-vertebral angle and along the vertebral column; but sometimes, the hand is prevented from reaching these deep-seated regions by the abdominal fulness consequent upon enlargement of the mesenteric glands. In many cases, it is easy to recognise the existence of ascites. Let me also remark, that at the very beginning of the disease, and prior to the cachectic state, persons have been supposed to be suffering from anasarca, caused by visceral affections, whereas, in reality, they had simply extensive œdema of the extremities, and ascites, caused by embarrassment of the general and portal circulations.

In only three of my eleven cases have I observed hypertrophy of the liver and spleen. There may be great enlargement of the latter organ. In the case of a woman, twenty-three years of age, I saw the spleen occupying the whole of the left side of the abdomen, extending to the umbilicus, and filling the iliac fossa. It is important to note that in this case there was no leucæmia.

I shall very soon revert to the consideration of the manner in which the symptoms of adenia are connected with one another; but before doing so, I wish to describe to you the case by which I have been most struck.

On February 20th, 1863, I was consulted by Madame X., aged twenty-three, affected with general glandular hypertrophy. At that date she looked in good health. Her temperament was not lymphatic: her colour was sufficiently good: her eyes were bright: and had attention not been called to her condition by the presence of greatly enlarged submaxillary glands, one would have been far from believing that she was the subject of any serious organic mischief. She had not suffered in childhood from any scrofulous affection, from suppurating glands, nor from chronic coryza: in her later years, she had had neither herpetic sore throat nor persistent sore eyes. This young woman had always menstruated regularly: she married at nineteen; and had two children who are in good health. There is a deficiency of information as to the antecedents of this patient's family; but we know that her father and mother are both living and in good health.

About nine months ago, that is to say, in the third or fourth month of her last pregnancy, Madame X. observed that she had small tumors in the groins and axillæ: in a fortnight, these glandular swellings acquired considerable volume, and other glands became enlarged, including glands at the angle of the jaw and in the occipito-cervical regions. The attention of Drs. Henrot and Landouzy (of Rheims) was arrested by so general an enlargement of the glands, and one which was so rapid in its progress. They attributed Madame X.'s cough to a similar enlargement of the bronchial glands. She had a favorable confinement. After the birth of her child, large glandular masses were detected in the iliac fossæ, and in the sacro-lumbar region. When the patient first came to me, a general glandular hypertrophy was apparent: the occipital, submaxillary, cervical, inguinal, epitrochlean, popliteal, and axillary glands were as large as eggs of pigeons or hens. On making digital pressure on either side behind the pubic arch, a deep chain of glands was felt. By palpation, the spleen was found to be much increased in size. This young woman never had had intermittent or hectic fever. I beg you to remark particularly, that her health always had been, and was when I examined her, unexceptionably good.

It being important to ascertain whether the blood contained an excess of white globules or of globulines, an examination was made by M. Dumontpallier. The result showed that it contained exceedingly few white globules, which were lost amid a great number of normal red globules: no globulines were found.

I have mentioned that the patient had a small dry cough. She never had had hæmoptysis. On the left side, respiration was natural; but on the right side, during expiration, there was a bellows sound and moist râles. There was no notable dulness at the apex of the lungs. The heart was healthy. There was no blowing sound in the vessels of the neck. There was no œdema of the extremities. There was no loss of appetite: digestion was easily accomplished: nutrition seemed normal; and there was no appreciable loss of flesh. Sleep was good: there was no depression of spirits. In fact, Madame X. would not have thought of making any complaint, had it not been for the annoyance she experienced from the formation of the glandular tumors.

Here then is the case of a young woman who had always enjoyed good health up to the third month of a pregnancy, when she perceived that a great number of small tumors were forming below the inferior maxilla, in the neck, axillæ, and groins, which tumors soon increased in number, and acquired a great size. She had a good confinement. She was able to nurse her infant for some months, after which her milk dried up; and then a new impetus was given to the development of the tumors. The lady's health, however, appeared satisfactory, up to the day on which she consulted me. She had a good complexion, experienced no pains anywhere, digested her food easily, and seemed to be normally nourished by it. The short dry cough by which she became affected during her pregnancy continued; but as I could not associate it with any serious lesion of the lungs, I adopted the opinion of Drs. Henrot and Landouzy: with them, I believed, that the cough was caused by pressure on the air passages occasioned by hypertrophy probably of the glands surrounding the right bronchus. This explanation was founded on the absence of organic lesions of the lungs, and on the generally increased size of the superficial and deep-seated lymphatic glands. I did not conceal from the family my uneasy forebodings as to the issue of the disease. There was, in fact, a probability that the patient would succumb to repeated attacks of suffocation, to asphyxia arising from bronchial obstruction, or to the cachectic state progressing so rapidly as to induce marasmus, profuse sweating, or colliquative diarrhœa. My sad forebodings were not long in being verified: the general modifying measures which I recommended were of no avail; and I learned, some months later, that the patient died from cerebral symptoms, having previously had sub-cutaneous hemorrhages.

So far as I know, no examination of the blood in the latter months of pregnancy had been made; and I have no grounds for absolutely denying that leucæmia existed in the latter stage of the disease. But if it be supposed, that the lymphatic glands and the spleen had acquired, after my examination, a very much greater development than that usually observed in cases of leucæmia, and that as yet leucocytes had not been in excess—if, again, we compare the case of this private patient with the cases we have now been studying, in which examination by the microscope never enabled us to detect excess of leucocytes—we are justified in supposing that the progress of the disease was that usually observed, and that, in all probability, the patient never had been leucocythæmic.

Notwithstanding this imperfection in the history, the case teaches the important lesson that a person apparently in perfect health, and whose blood seems in a normal condition, may be the subject of hypertrophy of all the lymphatic glands and of the spleen, and may sink very rapidly under the influence of glandular cachexia. If the patient be not leucocythæmic, are we to conclude that the blood has retained its normal composition? Certainly not: for in all the cachexiæ, the constitution of the blood is essentially modified: but the point I wish to establish is, that for at least the first ten months of her affection, the patient presented no symptoms of hæmatic lesion, nor of splenic or lymphatic leucæmia.

Once when commenting upon this case, I remarked to you, that should the patient not succumb from the cachexia, she might die asphyxiated from air not reaching the lungs. An example of this termination of general adenia I recollect occurring in the case of a young man whom I saw with my lamented colleague, the late M. Amussat.

More than twenty years ago, that distinguished surgeon summoned me to Néothermes to see one of his patients, a young man whose usual residence was at Poitou. For nearly a year, he had been the subject of general glandular engorgement. The lesion, however, existed more particularly in the neck, thence extending behind the clavicle and sternum. This condition produced so much difficulty in breathing that death seemed imminent. M. Amussat wished me to perform tracheotomy; but I said to him, that, although I was accustomed to perform tracheotomy in children having croup, and in adults having chronic diseases of the larynx, I felt that I ought in the case under discussion to leave to men of more authority, and to hands more practised, an operation beset with peculiar difficulties. I perceived that both the trachea and the blood-vessels were displaced, lost, as it were, amid numerous tumors; and that I could not introduce the bistoury with safety into regions where I had no guide in the anatomical relations of the parts.

M. Amussat, however, refused to perform the operation, fearing that affection for the patient, who was his intimate personal friend, might make his hand unsteady. Under these circumstances, it became incumbent on me to undertake the perilous duty, relying on the assistance of my friend who did not wish to undertake the principal part in the operation.

As soon as I had divided the superficial cervical aponeurosis, the tumors, till then imprisoned, spurted out, as it were, liberated by my incision, respiration, at the same time, becoming a little less difficult. It was then necessary for me to turn out with blunt hooks seven or eight tumors placed in front and at the sides of the trachea, which I exposed with extreme difficulty, having to avoid with the greatest care the numerous veins which presented themselves. Having opened the trachea, I introduced into it a large canula; but to my great regret, this hardly produced any alleviation of the dyspnœa: it was evident, that the passage, beyond the internal extremity of my canula, was compressed by other tumors. Having, however, dreaded this occurrence, I had provided myself with a canula wider than I generally employ in adults, and the length of which exceeded ten centimeters. This I substituted for the canula which I first employed. When I had introduced it nearly six centimeters, I came upon an obstacle which with difficulty, and by using an alternation of pushing and withdrawing movements, I was able to surmount. When once this obstacle was overcome, respiration became easy; but this did not prevent the patient dying on the following day.

For two reasons, I have been anxious to lay this case before you: first, to show you the manner of death when the bronchial glands and the glands in the course of the trachea are involved in the adenia; and second, to point out how little hope of success tracheotomy offers in these cases. In a patient from Stockholm, who came to consult me at another period, attacks of asphyxia sufficiently informed me that the trachea and bifurcation of the bronchi were constricted by glandular tumors.

This is the manner in which the fatal issue is hastened in patients affected with adenia. We see a particular patient die eight or ten months after the beginning of the disease, who might have lived several years longer, like the patient of M. Bonfils, and like several other patients whose history I have related, had the disease more specially attacked parts in which the consequent disorders could not become immediately dangerous.

I have just given you proof of the necessity of performing tracheotomy in certain cases of adenia. You have seen that opening the trachea only prolonged life some hours : as, however, our object was to avert death I should not hesitate again to recommend the operation, even though I were convinced that it was an almost desperate resource.

Let us now study the progress of the dyspnœa when the tracheal and bronchial glands are invaded. In the first stage of the disease, that is to say, before there is any cachexia, and when the hypertrophy of the glands has not reached this last limit, the patients often complain of oppression, of inability to walk quickly, and of not being able to go up a stair without being blown. Though this dyspnœa is organic, it sometimes shows itself with the characteristics of nervous dyspnœa. Generally, the patients are seized with dyspnœa during the night : they are unable to sleep in bed, and consequently pass their nights in an arm-chair. My Stockholm patient wrote to tell me that he felt a tightness in the respiratory tubes [*un resserrement dans les tuyaux respiratoires*]. One of the patients (an account of whose case has been communicated to me by Dr. Leudet) had had for some time shortness of breath and fits of dyspnœa. In both these cases, I was enabled, by attentive observation, to ascertain that there was no lesion of lungs or heart. The lungs may become accustomed to the pressure, and so the fits of dyspnœa may become less frequent : but in this case, either from rapid increase in the volume of the tumors, or from the enfeebled patient requiring at times to inspire a greater quantity of air, it was observed that in the latter days of his life, the suffocative attacks were more formidable, being accompanied by purple lips, haggard eyes, great anxiety, and cold extremities. The pulse became thready, and the inspiration hissing.

The embarrassed respiration may occasion passive hyperæmia of the lungs, causing mucous râles. Dr. Leudet has sometimes met with pleuritic effusion on one or both sides independent of any appreciable inflammatory action. Without seeking to explain this effusion into the pleural cavity, I ask :—Is this effusion, which does not originate in inflammation, analogous to œdema of the extremities induced by impediment to the circulation when the glands in the course of the vertebral column are themselves hypertrophied, as was found at the autopsy of one of the cases communicated to me by Dr. Leudet? When I come to speak of the pathological anatomy of the disease, I will relate to you this case of the Professor of the Medical School of Rouen. At present, as my more immediate object is to establish the symptomatology of this strange disease, I wish to describe two cases which occurred under my own observation, which show the frequency of dyspnœa in patients affected with adenia. No doubt can exist as to the mechanical cause of the dyspnœa in M. Amussat's case—as will be still better seen when we discuss the pathological anatomy of the disease.

On the 5th April, 1863, I sent back to Bonnevau a patient who consulted me for the first time in February of that year. He resided in the vicinity of Fontainbleau; and his ordinary medical attendants were Drs. Escalonne and Leblanc.

N., by occupation a wheel-wright, was 68 years of age. He had always enjoyed excellent health. In October, 1862, he perceived a small, perfectly indolent, tumor in the neck, to which he attached no importance. A month later, he had as it were an explosion of tumors in the neck, axillæ, and groins. When I saw him in February, 1863, three months and a half after the first appearance of the tumors, the neck was deformed by a multiplicity of round elastic tumors, which were blended with one another, or soldered together. The tumors in the axilla were specially remarkable for their size: those in the groins were not so large. I could not feel any in the abdomen. There was no hypertrophy of the spleen. The complexion was good; and the general health was excellent. I prescribed baths medicated with the bichloride of mercury, and the internal use of the tincture of iodine.

When I next saw the patient, I was struck with the increased size of the tumors. The neck was monstrous, being in circumference as large as the head, and having a frightfully lumpy appearance. In front of each ear, there was an enormous, vertically situated, long-shaped tumor, which was joined below to a circular band of other tumors occupying the submaxillary and lateral regions.

The face, fresher and more florid than is usual in an old man of sixty-eight, was, as it were, set in a frame formed by a mass of tumors occupying the neck anteriorly and laterally, and situated in front of the ears. At the base of the neck, and behind the clavicles, were numerous tumors embedded in the muscles, and extending into the chest. There were likewise engorged glands at the back of the neck, reaching to its base.

Under the axillæ, the swelling had quite a monstrous appearance. In each arm-pit, there was a tumor as large as the head of a full-term fœtus, looking like a large mamma, multilobular at its base, and resting on a mass of engorged glands.

In the groins, the tumors were smaller, though of considerable size. On palpation of the abdomen, numerous tumors were felt, particularly in the left side. The liver was slightly hypertrophied. The spleen was healthy.

The complexion was excellent, and the health was good; but respiration was difficult and whistling, and mucous râles were heard in the chest. The voice retained the normal tone and character, showing that the larynx had not undergone any morbid alteration. Compression of the trachea and bronchial tubes was probably the cause of the whistling respiration.

During the following month, I saw the patient for the third time. The glandular tumors had acquired a still more monstrous size. Although those in the anterior part of the axilla had acquired a size and aspect giving them the appearance of mammæ covered by a network of veins, I did not discover any œdema of the hands. There did not seem to be any loss of health, and the appetite had improved: nevertheless, it was easy to make the gums bleed. Neither the spleen nor liver were hypertrophied. The abdominal glands were more numerous and larger than in the previous month.

I recommended the patient to continue the internal use of tincture of iodine, and to take thrice a week sublimate baths. The pain and slight redness which I had observed in one of the tumors of the right axilla, led me to hope that these tumors might lose their hardness, and be absorbed. I knew, however, that adenia never terminates in acute inflammation and suppuration of the glands. At first, the patient found himself the better of the treatment which I had recommended; but I afterwards learned from Dr. Escalonne that the cervical and axillary tumors had acquired an extraordinary size, and that compression had rendered the trachea impervious to air. The patient died in a state of asphyxia, no other morbid condition having apparently been induced. The tumors did not suppurate.

The specially remarkable feature in this case is the sudden appearance of a multiplicity of tumors a month after a small indolent swelling had shown itself in the neck. Within three months, the submaxillary and cervical tumors had acquired so great a magnitude, that the base of the face, continuous with the neck down to the clavicles, was larger than the rest of the head. It was apparent, moreover, as is shown by my description, that these enormous tumors were continuous behind the clavicles and sternum: they accounted for the patient's dyspnœa and whistling respiration. In this particular case, the abdominal glands were numerous, and of very large size: the liver and spleen were normal. Notwithstanding this general and rapidly developed hypertrophy, the general health of the patient did not, at first, appear to be affected: it was not till the close of the fourth month, that there was bleeding from the gums. I regret that no microscopic examination of the blood was made at this period.

Some months later, I received, in my consulting-room, a gentleman from Stockholm, aged about thirty, who came for my opinion regarding tumors in various parts of his body. For a long time, he had had a running from the left ear: then, after having suffered during the whole of the year 1861 from a feeling of discomfort which he could not account for, he remarked, in June 1862, that a small tumor had appeared below the discharging ear. Three weeks later, several similar tumors became developed on the same side of the neck, and were not long in acquiring a great size. The right side of the neck, the groins, and the axillæ soon became the seats of tumors. Professor Malmsten (of Stockholm) recommended the baths of Kreuznach, which accordingly were taken in September 1862. After the course of baths, the debility became more marked, the sweatings were profuse, and the appetite nearly lost. The tumors, however, did not seem to increase in volume, and from about the end of December of that year to the 20th January 1863, they notably decreased in size: there was a perceptible return of the appetite, and, subsequently, of the strength. But after the end of January, the patient's appetite diminished, he became very impressionable, and his glands reassumed the size which they had lost. Every evening, there was a slight paroxysm of fever. Such was the condition of the patient when he came to consult me. The cervical and axillary glands were very large. I did not detect hypertrophy of the liver or spleen. I recommended sublimate baths and a tonic regimen. In the first instance, this treatment did not seem beneficial; for six weeks later, the patient wrote to me to say, that he continued in a state of great debility; that being unable to lie on his bed, he was obliged to pass his nights in an arm-chair; and that, as soon as he attempted to stretch out his legs, he was seized with oppression at the chest, and felt a sensation of constriction in the respiratory passages. In June of the same year, I learned that my patient had consulted Virchow. The learned professor declared the malady to be curable; and thought the adenia had originated in the previous discharge from the left ear. On this occasion, it was ascertained that there was no organic lesion of the lungs, heart, liver, or spleen. Virchow, moreover, affirmed that there was no leucocythæmia. With a view to soften the tumors, and facilitate their absorption, he recommended that they should be treated by local cold baths.

Here, Gentlemen, I must remark that, in this particular case, adenia was confined to the upper part of the body, there having been no hypertrophy of the glands in the inguinal and abdominal regions, nor of the liver or spleen. Virchow himself had affirmed that there was no leucocythæmia. The affection was, therefore, special in its character; and, although the learned professor of Berlin did not anticipate a fatal issue, he agreed with me in considering the glandular affection to be of a special nature.

In giving you the history of this case, I mentioned that the chronic discharge from the left ear was the starting point of the adenia. I will by-and-by make use of this etiological statement, connecting with it analogous cases in which acute or chronic local irritation—irritation of the nasal or ocular mucous membrane—seem to have played an important part. Perhaps, there is reason to suspect that in these cases there had existed some forgotten irritation of the skin or mucous membrane to account for the original adenopathy, the precursor of the outbreak of general adenia.

I have told you that the patients may sink from the severity of the suffocative paroxysms: I have endeavoured to point out to you the part which hypertrophy of the tracheal and bronchial glands seems to have in the production of those phenomena, admitting at the same time, that the nervous condition of the patient has some share in their production. Derangement of the functions of digestion, innervation, and nutrition, important in itself, though only of secondary importance in relation to the progress of the adenia, is sooner or later observed. In the second stage of the affection, there is loss of appetite: digestion is slow and painful: nevertheless, diarrhœa seldom occurs. Ere long, wasting supervenes, accompanied by great œdema of the extremities, and extreme debility. The hands are occasionally the seat of erythematous eruptions, and sometimes the legs are covered with ecchymotic spots, or the cutaneous affection may be cachectic pemphigus, as in the case of Dr. Leudet's patient. Some are exhausted by profuse sweating and hectic fever, as occurred in the cases of Dr. Perrin's patient,[1] and in my Stockholm patient.

It appears then, that adenia terminates by occasioning great disturbance of the principal functions: hematosis is soon interfered with. Adenia lowers the temperature of the body, or, to speak more correctly, it renders the patients more sensitive to cold. It disorders digestion. It induces cold sweats and hectic fever. It is, consequently, an affection of very great gravity, and one in which the physician has hitherto found himself pretty nearly powerless. The usual duration of the disease is from eighteen months to two years.

I am now going to read to you the report of a case observed by Professor Leudet, in his wards of the Hôtel Dieu of Rouen: it is very complete, and will enable you to study the progress of the disease and its termination in cachexia.

"Victor R., a tall man, aged fifty-seven, was admitted to the Hôtel Dieu on the 6th December, 1862. In former years, he had been exceptionally plump. When about eighteen years of age, he had begun to take alcoholic drinks freely: he was drunk at least once a week, and on the day following the debauch, could only eat food of high relish or dressed with vinegar. In his youth, he had never had engorgement of the glands, nor otorrhœa, nor ophthalmia. He had passed the whole of his military service in a regiment of cuirassiers: during that period, he contracted chancres followed by a suppurating inguinal bubo. No ulterior symptoms of constitutional syphilis were manifested by the skin, mucous membranes, or bones. After his discharge from military service, he took alcoholic drinks to still greater excess than formerly. The only morbid effects of these excesses were pains in the stomach, there being absence of vomiting, diarrhœa, jaundice, or derangement of the functions of the nervous system. The patient had had no serious illness prior to the adenia.

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[1] PERRIN:—*Bulletins de la Société Anatomique* for 1861, p. 247.

Four years ago, tumors appeared in rapid succession on the anterior and external aspect of both legs, and about the middle of the posterior surface of the right forearm: they were hard, did not suppurate, and were the seat of pain equally by day and by night. These tumors, of which the patient gave a very incomplete description, must have disappeared after two or three weeks of treatment, during which recourse was had to the iodide of potassium. He continued very weak for nearly six weeks after this illness, nevertheless, not experiencing any notable pain in the joints or limbs. This weakness entirely left him, and he never again experienced anything resembling it.

"Fifteen months ago, chronic coryza appeared, with loss of the sense of smell, but unaccompanied by any feeling of general discomfort: some months after the beginning of the coryza (which was purulent and somewhat sanguinolent), an affection was observed at the inner angle of the left eye, in the situation of the lacrymal sac. This affection, which terminated in suppuration and cicatrisation, must have occurred after the illness had lasted three months. It was during the last stage of the inflammation of the lacrymal sac that R. observed, for the first time, that the lymphatic glands of the neck were swollen. His strength became at the same time greatly enfeebled, without there being any paralysis.

"Ten months ago was the date of his first admission into the Hôtel Dieu. He was received at that date into the surgical department, for conspicuous swelling of the lymphatic glands of the cervical, axillary, and inguinal regions. R. remained five months and a half in the surgical wards. During that period, the glands decreased in size, were devoid of pain, and presented the same characters as at my first examination. During the same period, he experienced no disturbance of the digestive organs, nor had diarrhœa, epistaxis, or any kind of hemorrhage.

"On leaving the surgical wards of the Hôtel Dieu, on the 25th October 1862, he was employed in the charity work-shops, living on seventy-five centimes a day. During that period, he experienced great loss of strength. About the middle of November, 1862, he became affected with cough, accompanied by pain in the right side of the chest.

"When admitted into my wards, I found R. in a state which I shall now describe to you:—The face was pale: there was still a certain plumpness of appearance, though, according to the patient's statement, that had much diminished. There was a marked depression of vital power, particularly since the cough had set in. There was anorexia of recent date. There was no diarrhœa. The face was swollen, particularly in the submaxillary and subhyoid regions, in which, by palpation, could be recognised twenty round, moveable tumors, which were not painful even when pressed. The most bulky of these tumors were as large as pigeons' eggs, and the smallest were the size of filbert nuts. The hypertrophied glands gave the appearance of a double chin, with lateral enlargement of the face. There were tumors of similar character in the subclavicular regions, but none in the back of the neck. The largest tumors were in the axillæ, particularly in the right axilla, where the aggregate bulk exceeded that of an adult's fist: it was made up by other smaller tumors which were not fused together. There was a similar but not so great an enlargement of the glands in both inguinal regions, and in the inguino-femoral triangle. Several small enlarged glands existed above the condyles on both sides. The enlarged glands were nowhere adherent to the skin, which presented neither change of color nor vascular dilatation.

"The coryza continued, retaining its purulent and sanguinolent character. There was no deformity of the bones of the nose. The skin was adherent, in the situation of the left lacrymal sac, which adhesion at the inner angle caused depression—to the extent of one half—of the left upper eyelid. There was no redness of the eye; and vision was unaffected.

"For three weeks, the cough had been very severe; the expectoration was slightly muco-purulent, devoid of brown colour, and yielding a fetid smell like that of sphacelus. By auscultation, numerous subcrepitant and even some sibilant râles were heard on both sides of the chest: they were more numerous on the right than on the left side. There was slight tension of the abdomen, but no ascites. In consequence of the abdominal walls being capable of only slight depression, the detection of enlarged glands within the abdomen was impossible. There was no morbid increase of spleen or liver. The urine did not deposit a sediment: on examination by heat and nitric acid, it gave no indication of albumen; nor did it yield any trace of glucose on being tested by potassa and Barreswil's solution. The blood, which was examined several days after the patient's admission to hospital presented nothing abnormal—there was no numerical augmentation of the white globules or globulines, and the red globules were normal. That examination, repeated several times between that time and the present date (7 April 1863), has always yielded similar results. I prescribed Baréges water, twelve drops of essence of turpentine in a julep, weak wine and water as a drink, and ordinary diet.

"During December 1862, R.'s condition ameliorated, the fits of coughing became less intense, and never occasioned real orthopnœa: the fœtid odour of the sputa diminished, and finally disappeared about the end of that month. The strength was always below the natural standard. There was no diminution in the size of the glands. About the end of December, slight œdema supervened in the lower extremities. The urine continued to be non-albuminous. I ordered decoction of cinchona, pills of the iodide of iron, wine and water as a drink, and a double allowance of aliment and of wine.

"During January 1863, there was a slight increase in the size of the glands, particularly in those of the neck and axillæ: there was some enlargement of the abdomen, and the inferior margin of the liver began to be appreciable below the false ribs: there was an extension of the splenic dulness, but the inferior margin of the spleen could not be felt below the left false ribs: there was a little increase in the œdema of the lower limbs. The bronchitis was nearly at an end: some sibilant and subcrepitant râles were heard at the base of both lungs. The purulent discharge from the nose continued. There was no longer any fœtid expectoration. The patient improved somewhat in strength: he was able to be up during the greater part of the day: and his only complaint was of the inconvenience caused by the œdema of the legs and the abdominal swelling.

"From the middle of February 1863, to the beginning of April, the lymphatic glands in the submaxillary, subclavicular, axillary, subepitrochlean, and inguinal regions, in succession, increased in size, but preserved the same characteristics which they presented when the patient was admitted to the hospital. Day by day, there was a marked increase in the volume of the spleen, which was observed to have descended to the anterior superior spine of the ilium, its sharp edge being apparently directed forwards. Ascites existed to a slight extent. There was never any symptom of cardiac disease. The liver extended, at the least, two finger breadths beyond the margin of the right false ribs. The patient's strength was good;

and he occupied a part of each day in walking in the court. He continued under careful observation in my wards.

"From June 1863, R.'s state became more serious; and there was a return of the diarrhœa. At the beginning of that month, the lower extremities became the seat of slight œdema, and some bullæ of cachectic pemphigus appeared on the dorsal aspect of the carpus of one hand. The ozœna continued. From about the middle of June, there was a continuance of slight dyspnœa, increasing when he moved, but without any roaring [*cornage*].

"At the beginning of July, the debility had become more marked. At this date, there was no albumen in the urine, *nor any leucocythæmic alteration of the blood*. On the 15th July, I detected pleuritic effusion occupying the lower half of the right pleura. Death occurred on 19th July, without there having been any dyspnœal paroxysm or alteration of voice.

"An *autopsy* was made on the 21st July, thirty-seven hours after death.

"The cranium and brain presented no morbid appearances. The nasal fossæ (examined behind only, from the body being claimed) presented marked thickening with a greyish tint and softening, without ulceration of the membrane. The bones, examined by the cranium, seemed to be in a perfectly healthy state.

"There was no morbid change in the larynx, trachea, nor in either bronchus: there was neither flattening of their parietes, nor change in their calibre.

"In the lower half of the right pleural sac, there was about half a litre of effusion composed of a gelatinous serosity tinged with blood. A layer of soft citrine-colored serosity covered the rest of the lung. This lung was rather more resistant than a normal lung—somewhat hard as in non-granular pneumonia. There was no deposit of morbid tissue. The bronchial tubes were not dilated. There was hardly any engorgement of the left lung. The heart and pericardium were healthy. The pulmonary artery was free from embolism.

"There was a little effusion into the peritoneal cavity—about half a litre of citrine colored serosity free from false membranes. The liver was normal in size, yellowish in color, without manifesting this hue externally: it was in a state of fatty or amylaceous degeneration. The spleen was much enlarged. Its length was $0^{m}\cdot 26$, and its breadth $0^{m}\cdot 17$. There was no thickening of the fibrous capsule. The parenchyma had a color like that of the lees of white wine, was not soft in consistence, nor had it any thickening of the stroma. The kidneys were of the usual size, somewhat pale, and healthy in structure.

"There was great enlargement of the lymphatic glands, particularly in the neck, submaxillary and subclavicular regions, in both axillæ, in the abdominal cavity in front of the vertebral column, and in both inguinal regions. Most of these enlarged glands were collected and weighed together, when their weight was found to be a little under four kilogrammes. They varied in size: the largest were those of the abdomen, whether taken singly or collectively. In that region, they formed a mass nearly as large as the head of an adult. In the axillæ, the glandular masses were the size of a young person's fist. The largest of the enlarged glands—those of the abdomen—were the size of a turkey's egg. The cervical, axillary, and inguinal glands were redder than those of the abdomen, which had a whitish hue. The capsule was not thickened; and the contained gland was throughout rather soft, diffluent, and without thickening of the stroma.

"There were also some hypertrophied glands in the fold of both arms. A slightly flattened substance, formed by rather voluminous reddish glands existed in both vertebral sulci, below the parietal pleura.

"The bronchial glands were relatively much less developed than those of the neck, and particularly than those of the axillæ: two of them, however, were as large as filbert nuts, but did not compress the vessels or air tubes.

"The thoracic cavity was healthy.

"The microscopic examination of some of the glandular tumors showed me that their parenchyma contained nothing cancerous, nothing fibro-plastic, and nothing of the nature of amylaceous degeneration, but only minute nuclei, less than the elementary cells of lymph, and somewhat resembling epithelial cells."

To sum up the case:—A man, of fifty-seven years, a man who had always enjoyed good health, notwithstanding the frequency of his alcoholic excesses, and who in childhood had given no indication of scrofula, presented, four years ago on the legs and right forearm, tumors which terminated in suppuration, and might be regarded as syphilitic gummi. When this patient was admitted to Professor Leudet's wards, he was suffering from chronic coryza, and bore the cicatrix of a lacrymal tumor. It was during the suppuration of this lacrymal tumor, that the first cervical tumors appeared; and it was not till a later period that similar swellings became developed in the subclavicular, subaxillary, and inguinal regions. These tumors were large and indolent. Afterwards, the adenia progressing, and there being no doubt as to its generalisation, the increased size of the liver and spleen was detected. On several occasions, the blood was examined without the discovery of an excess of leucocytes or globulines; and there was an entire absence of the principal symptoms of splenic or lymphatic leucæmia. Four months before death, the glandular and splenic hypertrophy made rapid progress: the patient became more and more feeble, the œdema of his lower extremities more marked, and his ascites more considerable. At a later period, bullæ of cachectic pemphigus appeared on one of the feet: cough set in; and there was pleural effusion occupying the inferior third of the right side of the chest.

The autopsy showed that the serious lesions were confined to the spleen and lymphatic glands. It was also seen, that these lesions depended on simple hypertrophy of the organs, microscopic examination disclosing nothing more than an increase of normal elements.

However, although to ordinary methods of investigation, the blood presented no appreciable alteration, it was impossible to get rid of the belief that there existed in the case I have now detailed, and in other similar cases, a special dyscrasia of the blood; for the patients sunk into a state of cachexia, when not carried off by a suffocative paroxysm. This dyscrasia, with the nature of the essence of which we are not acquainted, is not a matter of doubt with me; and although general adenia produces no excess of leucocytes or globulines, nor diminution of the normal number of blood globules, it probably modifies the lymph elements in such a manner that at the end of a period of indefinite duration, anæmia sets in, which is followed by cachexia. English physicians, more particularly Dr. Pavy and Dr. Wilks, have described a variety of anæmia to which they gave the name of *lymphatic anæmia*, meaning to express by that term the lesions of glands which a special kind of anæmia may induce. I confess, Gentlemen, that I am disposed to accept this view.

In the present day, every one admits that the formation and maintenance of the blood are functions performed by the co-operation of certain organs: pathology, better even than physiology, proves the hematopoietic agency of these organs. We know, for example, that chronic affections of the liver and spleen produce serious consequences in respect of the composition of the blood; for whatever be the nature of these affections, they are followed by anæmia with modification of the number, consistence, form, color, and chemical composition of the blood globules. Tuberculosis of the mesentery leads to the same result: so does scrofula, the different manifestations of which have their principal seat in the lymphatic system.

Need I remind you of the influence of organic lesions of the lungs upon the composition of the blood? The necessary consequence of all these lesions is to interfere with the exchange of gas, the very essence of pulmonary hematosis, and so lead to anæmia. There is, then, a kind of anæmia which is of pulmonary origin, just as in all probability, there exists in the fœtus an anæmia of placental origin, when the placenta has undergone a partial fatty and fibrous degeneration. If then, we grant that there is a pulmonary anæmia, a splenic anæmia, and a hepatic anæmia, why should not we also accept a *lymphatic anæmia*? To be satisfied that it exists, we only require to bear in mind the function of the lymphatic system. The ramifications of this system, in the interior of organs and in the intestinal mucous membrane, derive the elements required for the maintenance of the blood. The lymph and the chyle undergo a modification in the parenchyma of the glands, and then all the lymphatic fluid, whatever may have been its origin, is poured into the venous system, and like the subhepatic, splenic, and intestinal blood, it passes on to the lungs, there to undergo such modifications as are necessary to convert it into blood suitable for the nutrition of all the organs. Gentlemen, I cannot tell you what special action on the composition of the blood is caused by general hypertrophy of the glands: I do not know whether it notably diminishes the leucocytes, but I am certain that it does not increase them. Microscopic investigation and chemical analysis have not informed us how the blood is modified in adenia, but the modification itself is demonstrated by the symptoms; and it cannot be otherwise when all the glands in the body have become greatly hypertrophied. Again, the modification of the blood is proved by the anæmia and cachexia under which the patients succumb when they do not die from asphyxia caused by compression of the trachea or bronchi. I hold then with Drs. Pavy and Wilks, that there is lymphatic anæmia, just as there is hepatic anæmia and splenic anæmia. Possibly, leucocythæmia, on which I have already lectured to you, is only a variety of anæmia characterised by an excess in the proportion of leucocytes or globulines. We know, in fact, that the presence of leucocytes, is compatible, in a certain measure, with health, and is met with in a great number of morbid states, without appearing to aggravate them: on the other hand, we know, that those organic lesions which are regarded by Virchow and Bennett as the causes of leucocythæmia may be wanting; and, finally, we know that hypertrophy of the lymphatic glands and hypertrophy of the spleen—the condition of organs peculiarly characteristic of the lesion—may exist, and often do exist in cases in which there is no leucocythæmia. It appears, therefore, that the only symptoms which declare the gravity of the malady are anæmia and cachexia, the latter being always the ultimate consequence of deep and long continued anæmia.

Clinical observation has told us that the malady is characterised by an augmentation in the size of the glands: necroscopic examination has absolutely proved that the tumors are in the lymphatic glands, and that the surrounding tissues do not present the least trace of inflammatory action. By careful dissection, we can isolate each of the individual glands which in the aggregate constitute the glandular masses. These tumors—mammary in form and elastic in consistence—have sometimes attained an enormous weight. Dr. Bonfils met with an inguinal tumor of this kind which weighed 2,250 grammes, the axillary tumors weighing 1,000 and 500 grammes. The superficial and deep cervical glands, the occipital glands and the whole circum-maxillary chain of hypertrophied glands gives the face a peculiar appearance: they may vary in size between a pigeon's egg and a hen's egg. The deep-seated glands—that is to say, those in the thoracic and abdominal cavities—may become greatly hypertrophied. In the chest, the tumors are seldom larger than a pigeon's egg; but from their disposition around the trachea and bronchi, they may compress these organs. At no autopsy has it ever been found, that the glandular masses compressed and deformed the large vessels near the heart. The intra-abdominal glandular masses are found to be constituted by the pelvic, lumbar, and aortic glands; and by the mesenteric, mesocolic, gastro-hepatic, gastro-splenic, gastric, and pancreatic glands. In the case of Dr. Bonfils, the former (which belong to the general lymphatic system) weighed 3,620 grammes; and in Dr. Leudet's case, some abdominal glands were as large as turkeys' eggs. The glands of the chyliferous system usually constitute less voluminous tumors: Dr. Bonfils, however, has observed them sometimes as large as pigeons' eggs. Let me add, that the glandular masses in the axillary, and submaxillary regions are sometimes continuous with the intra-thoracic masses by means of bands of hypertrophied glands. A similar remark is applicable to the pelvic and inguinal glands. The chyliferous system is usually less affected, as I have already said, than the general lymphatic system: the glandular hypertrophy may also, however, have its seat in the glands of Peyer, as was found in Dr. Potain's case.[1] "From about the middle of the small intestine to the ileo-cæcal valve, a great number of the solitary glands were seen to be white and prominent: Peyer's patches to the number of twenty were very apparent, slightly prominent, a little granular on their surface, elastic, not notably hard, of a dull white color." It must also be stated that in this case some of the mesenteric glands were as large as hens' eggs.

My object in entering into these anatomical details is to show you, that no gland escapes, and that in all of them the hypertrophy may be great.

All observers concur in stating that the envelope and stroma of the gland do not undergo any change. Section of the parenchyma presents a greyish appearance in the smaller glands, a yellowish grey in those of medium size, and a yellowish color in those of larger volume. The latter alone present ecchymotic spots in their interior; and their section, when scraped, yields a turbid whitish fluid which mixes with water: one might suppose that they contained a fluid analogous to that of cancer containing large cellules, or nucleoli; but such is not the case: MM. Charles Robin, Leudet, and Potain have all stated, that the glands do not contain any of the elements called cancerous but only nuclei and lymphatic cells heaped up one on another—that is to say, that in adenia the glands increase in size, but the connective tissue of the gland retains its natural disposition: there is only a hypergenesis of cells and lymph nuclei, which cells and nuclei, nevertheless, are perfectly normal. Such was the conviction of Dr. Laboulbène and Titon when they studied the structure of the glands of a patient, who, in 1852, was admitted into the Hôpital Sainte-Marguerite, under the care of Dr. Marrotte, to be treated for adenia, an affection, which at that period, Dr. Laboulbène designated in his notes as "general adenitis" [*adénite généralisée*].

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[1] Potain:—*Bulletin de la Société Anatomique*, 1861, p. 220.

Up to that date, no one had injected the afferent and efferent lymphatic vessels: injection would probably have shown some interesting peculiarity in the rich parenchymatous network, and in its permeability.

Hypergenesis of the cellular elements of the gland is then, it appears, the principal anatomical fact in adenia: it is the starting fact, the origin, so to speak, of all the secondary lesions. These secondary lesions, however, ought to be more particularly studied in the spleen and liver. Hypertrophy of these organs is hardly observed till the second stage of the disease. The spleen, especially, may acquire a great size: as you have seen, that organ may occupy the whole of the left side of the abdominal cavity, and descend to the pubes. In twelve cases of adenia which I analysed, in four only was the spleen increased in size. In the case of Dr. Bonfils, it weighed one kilogramme, and measured 24 centimeters in length by 15 centimeters in breadth. In the case of Dr. Leudet, it measured 26 centimeters in length by 17 centimeters in breadth. In the examination made by Drs. Bonfils and Leudet, there was no observable structural modification; but in Dr. Potain's case, although the spleen scarcely exceeded the normal volume, its section presented a uniform bright red color exhibiting the cut surfaces of very many *white* points of the size of hemp seed, and very apparent whitish trabeculæ. The white points presented granules of a slightly irregular rounded shape, of feeble consistence; and which were constituted by nuclei in all respects similar to the nuclei found in the lymphatic glands.

In no case did the liver present any remarkable alteration. Generally, nothing more was discovered than slight hyperemia of the organ, without any alteration of capsule, stroma, or hepatic cells.

No microscopic or chemical analysis was made of the contents of the receptaculum chyli, nor thoracic duct, the great lymphatic trunk of the right side. It would have been important to have discovered whether, in these different localities, there was any diminution or augmentation of the lymph globules, or any alteration in their form and color. Be that as it may, observers have not noted, *post mortem*, any modification in the blood found in the heart and great vessels. Microscopic examination instituted at the beginning of the last stage of the disease, has established the non-existence of leucocythæmia. Hence, it is exceedingly probable, that there is no increase in the lymph globules passing into the superior vena cava; and consequently, that the lymphatic ducts leading to that vein do not contain these globules in excess: it is even possible, that they are much less numerous than in the normal state.

The conclusion from all these facts is that, although the affection has its seat almost exclusively in the lymphatic glands, it is only in some exceptional cases that the intestinal glands share in the cellular hypergenesis constantly met with in the lymphatic glands. I ought, however, to mention that, in a case of adenia, reported by Dr. Hallé, which occurred in the hospital practice of M. Nélaton, the liver was studded by innumerable small white bodies cancerous in their general appearance, and varying in size between a lentil and a hazel nut. The spleen, which was enormously enlarged, presented, we are told, a great number of soft cancerous masses, of the size of a walnut, the white color of which contrasted with the red of the tissue of the spleen. It is not stated that the white masses found in the liver and spleen were demonstrated to be cancerous on microscopic examination, so that we are entitled to doubt that they were of that character. This doubt is the more allowable, as similar masses found in the spleen of Dr. Potain's patient presented, under microscopic examination, a structure identical with that of the lymphatic glands of the same patient. I am inclined to believe, that a similar statement is applicable to the so-called cancerous masses met with in Dr. Hallé's case.

Gentlemen, we have now reached the most interesting part of the subject of adenia—its *etiology*. Struck by the general affection of all the lymphatic glands, the first hypothesis naturally presented to the mind is the existence of a diathesis. Can we associate the lymphatic affection with any known diathesis? Can we cite in explanation of the general hypertrophy of the glands, the constitutional diseases termed scrofulous, tuberculous, cancerous, and syphilitic? Certainly not; for in infancy, the patients presented no manifestation of the scrofulous diathesis, and the glandular affection was developed at an age when scrofula has lost all its power. Moreover, to prove that neither the scrofulous, cancerous, nor syphilitic diathesis has anything to do with causing the disease which I call "adenia," it will be sufficient to study the glandular affection as a local affection.

Syphilis itself does not explain in a more satisfactory manner the cellular hypergenesis of the lymphatic glands; for if it be true that in secondary pox, the glands may be the seat of chronic inflammation, the inflammation never extends to more than a number of glands; and further, only one patient—Dr. Leudet's patient—had any syphilitic antecedents, and none of the other patients, including the young woman whose case I have reported to you, ever showed any syphilitic manifestation. Let me add, that treatment by mercurial preparations or iodide of potassium, though it may cure the syphilitic gummata as in Dr. Leudet's case, exerted no action on that man's glandular system; nor on that of any of the other patients, who from deficiency of exact information, and in default of knowledge of the cause of the disease, were subjected to that medication.

We are thus constrained to conclude, that there is a new *special* diathesis, the essential nature of which is unknown, which we call the *lymphatic diathesis.* This diathesis may be described as a tendency in certain persons to present, under the influence of a determining cause, glandular engorgements, at first local, and becoming general in from eighteen months to two years. This glandular engorgement, as I have seen, may consist in a hypergenesis of the normal cellular elements of the lymphatic glands, a hypergenesis which in some cases may invade the glandular corpuscles of the spleen and intestine. The patient, consequently, has anæmia and cachexia, unaccompanied by leucocytosis.

Adenia, I have said, is a diathesis which has a determining cause. What is this cause, and what is its most common seat? When we attentively peruse the reports of cases of adenia, whether described by others or observed by ourselves, we are struck by the fact that, in the first instance, only one or two glands have been enlarged: some weeks, or, it may be, two or three months after the appearance of these swellings a veritable explosion of glandular tumors occurs in different parts of the body, while, at the same time, the original tumors rapidly increase in size. In the majority of cases, the submaxillary are the glands which first become affected: sometimes, however, the first seat of the affection is in the axillary or inguinal glands.

Whenever there is an acute or chronic engorgement of glands, we must search in the regions which they depurate for some organic lesion to explain the glandular irritation. This rule, which is absolute, will be found to lead to many important results. It is natural, therefore, in a case of general adenia, to inquire, what local lesion has occasioned the original engorgement. There are many cases, however, in which no light is thrown upon this question: we must be satisfied to note that the engorgement commenced in the axillary, inguinal, or maxillary glands—which is provoking. Viewed along with these incomplete cases, there are others—the cases of Leudet, Potain, and Perrin, and the case of my Stockholm patient—which possess great interest in relation to this question. I have thrice observed that there existed acute or chronic irritation at the great angle of the eye or in the external auditory passage: and observe, Gentlemen, that the glands first attacked were situated on the same side as the ocular, nasal, or aural lesion, and that the submaxillary and cervical glands of the opposite side, as well as the other glands of the body, were only secondarily attacked. It is, therefore, well worthy of remark that, in the five cases to which I have referred, there were four with inflammatory lacrymal tumor, chronic coryza, and otorrhœa. One cannot help being struck with this alteration of the skin and mucous membranes, and with the primary glandular alteration. I ought, however, to remind you that, in one of Leudet's cases, and also in the case for which we are indebted to Perrin, the patients stated that the glandular engorgement began in the axillary region. Subsequently, however, MM. Leudet and Perrin discovered submaxillary engorgement, so that we may suppose the possibility of that engorgement having existed at the commencement of the adenia, but to so small an extent as not to attract the notice of the patients.

Be that as it may, it is a fact that, in twelve cases of adenia, there were four in which there existed lacrymal tumors, a chronic coryza, and an otorrhœa.

It is not a matter involved in the least doubt—it is a positive fact—that there is a relation between the primary adenopathia and the superficial lesions of skin and mucous membrane.

As to general consecutive adenia, I cannot understand, admitting certain persons to have a predisposition of such a special nature, that one or two lymphatic glands being engorged for a certain short period, in general of variable duration, but nearly always of recent date, should be the starting point of the generalisation of the malady to the other glands.

I have already told you the effect which adenia produces upon the composition of the blood. The microscope has shown that both before and after death, it contains no excess of white globules or globulines. And although there is no proof that the *quality* of the globules has undergone any modification, the characters of the adenia, at least in the second period of the disease do not leave the smallest room for doubt as the *quantity* of the red globules. It is probable, therefore, that in adenia, there is a diminution of the components of the red globules; and that this diminution is very probably the result of general enlargement of the glands.

It has also been observed by Wunderlich that, in a certain number of cases, the adenopathia does not become general till after it has been some time exclusively localised in a few glands. At other times, the affection begins with a general outbreak. Virchow is not in the slightest degree perplexed as to the secondary nature of the generalisation of the malady: by his favorite embolism, he explains the metastasis. The explanation of primary generalisation is not quite so simple. Moreover, Virchow's metastatic embolism explains nothing in respect of the special affection, because all those—and they are fortunately many—who have enlarged glands have not general adenopathia as a sequel. If the generalisation be the exception, it is because it is dependent on exceptional causes. For want of a better name, I call these exceptional causes *specific* causes.

Wunderlich (much more the clinical physician than Virchow) is of opinion that adenopathia, whether it become general on the first manifestation of the disease, or consecutively, is one and the same thing; and that it is a constitutional affection, just as cancer, whether it be general at its first outbreak, or do not become so till a subsequent period, is in both cases a constitutional affection. This statement does not imply that Wunderlich and I are acquainted with the essential nature of adenia. Is there anything of which any one knows the *nature?* It is only *effects* that are known.

But Wunderlich goes further: considering that there is no difference, except in the state of the blood, between leucocythæmia and adenia (which he calls pseudo-leucocythæmia) he asks—Whether adenia be not an early stage of leucocythæmia; and whether the difference between the two affections does not consist in a diversity of accessory causes, by which there is produced a great quantity of white globules in one case, and no white globules at all in the other case? Wunderlich explains the nature of this accessory cause: in leucocythæmia, he says, that there is hyperplasia of the interstitial connective tissue in the stroma of different organs, such as the liver, spleen, kidneys, and intestines; and as the lymphatic vessels originate in the plasmatic network of the connective tissue, the cellules, as they multiply, may enter the lymphatic vessels, and so get into the blood. This would explain the profusion of white globules in leucocythæmia, in which we find these conditions.[1]

I confess that till more ample information be obtained, I shall continue to look on leucocythæmia and adenia as two distinct affections, although very nearly approaching one another in respect of their lesions. To my mind, the fact of the progress of the disease being very different in the two cases is decisive of the question.

Does the study of adenia in which we have now been engaged furnish practicable therapeutic indications? Hitherto, no system of treatment which has been employed seems to have yielded a satisfactory result: it is well to state, however, that in three cases, the waters of Kreuznach and Lavey, and sublimate baths appeared, at least temporarily, to retard the progress of the disease. In the first case, the German waters reduced the volume of the engorged glands. A patient for whom I prescribed sublimate baths, did not, for some months, appear to suffer in his general health from the morbid alteration of the glands. M. Cossy reports the case of a patient, aged 53, affected with general hypertrophy of the glands, without leucocythæmia, whose condition was remarkably ameliorated by a two months' course of the waters of Lavey. In this particular case, however, the treatment was complex; for the waters were given internally to an extent sufficient to produce slight purging: hot and cold douches were played upon the glandular tumors as well as on the body generally: the tumors were also regularly subjected to shampooing: and the patient took daily three of Blancard's iodide of iron pills.

This treatment—on several occasions temporarily diminished in activity—led to amelioration, which began to show itself after the thirtieth day. The treatment was continued for two months; and when the patient left Lavey, there was diminution, to the extent of one half, in the volume of the engorged glands. In this particular case, resolution was more attributable to the physical process of applying the mineral waters than to the iodides and chlorides which they contain in small quantity, and which they impart to the mother water of the saline waters of Bex, with which they are generally mingled.

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[1] Wunderlich:—Pseudoleukæmia, Hodgkin's Krankheit, oder Multiple Lymphadenome ohne Leukæmie. [*Archiv der Heilkünde*, 1866, *p.* 531.] See also:—Paul Spillmann in the *Archives de Médecine* for August, 1867.

If in the case now related, the amelioration ought in part to be attributed to the action of the iodides and chlorides, it would perhaps be advisable to prefer to the waters of Kreuznach and Lavey those of Saxony, which (without the addition of the mother water) contain, according to Dr. Aviolat, the enormous quantity of thirty-three grammes of iodides and ten grammes of bromides in a bath of three hundred litres.

The general indications of treatment are furnished by the state of the glands, and the anæmia. Whenever we find primary engorgement, and recognise its cause, we ought to use every means at our disposal to remove the primary irritation; but when there are already numerous glands manifestly engorged, we must not hesitate to endeavour to modify the general morbid state of the patient, either by giving repeated saline purgatives, or mineral purgative waters, such as those of Kreuznach, Levay, and Saxony, or other waters of similar character. It is necessary at the same time to employ local treatment by douches and shampooing, to induce absorption of the superficial tumors. Finally, preparations of iodine, iron, and cinchona, will have the double advantage of modifying the general state of the system, and combating the anæmia which is apt to make rapid progress. I have, I confess, Gentlemen, only a moderate amount of confidence in this complex treatment of a disease which seems to me to have a specific character; but it is our duty, when we are without a specific remedy for a specific disease, to use every effort to counteract the disease by other medicines, even when it must be admitted that they only influence symptoms.

Gentlemen, I must not omit saying a few words regarding a glandular affection which I have very often observed in young Creoles, and particularly in Creoles from the Mauritius and the Island of Reunion. I cannot, however, be sure that it is not due to mere chance that I have seen this particular form of adenia in young persons born in the two colonies I have named, and never once in persons born in the French or English West Indies.

In adolescence, and more frequently in boys than in girls, we see the superficial and deep inguinal glands become swollen, sometimes only on one side, and at other times, simultaneously on both sides. The disease makes its appearance in paroxysms, lasting for one, two, or three months, separated from one another by intervals, it may be of several months. An attack of greater violence than its predecessors then sets in, and some of the glands suppurate. In certain cases, the suppuration extends to several glands, and in succession to the entire mass of glands. The patients are consequently condemned to a long confinement to bed or bed-room. Sometimes, the suppuration goes on for a year. It sometimes happens, very seldom, fortunately, that perinephritic abscesses form. This occurred in a case of which I shall have to speak when I come to treat of that subject. There may be a band of glands suppurating from the groin up to the kidney, with large abscesses forming round that organ. You can appreciate the danger involved in such an occurrence.

In the majority of cases which have come under my observation, the disease has ceased at the age of manhood without my being able to attribute much benefit to the medical treatment employed.

LECTURE XCII.

AMENORRHŒA AND MENORRHAGIC FEVER.

Menorrhagic Fever.—Amenorrhœa from Change of Residence does not call for any treatment; or at least there are no special indications of treatment.—Menstruation consists of two parts; viz. Ovulation, and Hemorrhagic Flux from the mucous membrane of the Fallopian tubes and Uterus.—Amenorrhœa from Chlorosis and from Anæmia.—Amenorrhœa, consequent upon Disease, Acute or Chronic.—Therapeutic Indications derived from the state of the General Health.—Therapeutic Opportunity.—General and Local Bloodletting: Hot Baths: Iodine: Emenagogues.

Gentlemen:—An error in diagnosis, which you saw me commit the other day, affords me an opportunity of entering into some details regarding an affection to which I give the name of menorrhagic fever [*fièvre ménorrhagique*].

A young woman of 17, who had been ill for six days, was admitted as a patient to Saint-Bernard's ward. She was born in the country; and had only resided four months in Paris. Like most new-comers, she did not menstruate. From the commencement of her indisposition, she complained of headache, giddiness, and insomnia. She had epistaxis, loss of appetite, foul tongue, and diarrhœa. The pulse was febrile; and there had been no cessation of fever. I concluded that the case was one of dothinenteria. On the following day, the catamenia appeared, and the fever abated. The menstruation was normal; and two days after the appearance of the sanguineous discharge, health was completely re-established. Gentlemen, this is not the first case of the kind which has presented itself in our wards: not a year passes without my calling your attention to such cases, of which sometimes I am led to form an erroneous diagnosis.

When the menstrual function takes place every month in a regular manner it is generally attended by discomfort of unimportant character; there are, nevertheless, headache, and a modification of the different functions which remind us of the febrile disturbance caused by slight indispositions: this arises from monthly ovulation being pathological to a certain extent, the turgescence of the ovaries and uterus, and rupture of the Graafian vesicle, constituting a sort of morbid process affecting some individuals more than others.

In many women, as you know, there supervenes not only the discomfort to which I have referred, but likewise symptoms of real fever: this is not to be wondered at when we reflect upon the individual peculiarities so frequently met with in practice.

Some persons, with a slight sore-throat, a boil, or an inflamed superficial gland, have violent fever, even sometimes accompanied by delirium. It is, therefore, not in the least surprising that, in exceptional circumstances, the process of ovulation should be accompanied by pretty severe febrile paroxysms.

We know, likewise, that the economy, be it ever so excitable, becomes quite habituated to morbid impressions often repeated in a similar manner. If, however, the same morbid impression recur after a very long interval, it is much more severely felt: on the other hand, in respect of menstruation, the phenomena attendant upon congestion and hæmorrhage are in general aggravated in proportion to the length of time during which the function has been suspended. There exists then a twofold reason for the menorrhagic fever being more violent.

Having been led to discuss this subject, I must not neglect the opportunity of speaking to you of amenorrhœa and its treatment. You have seen me so often employ very different forms of treatment, that you must naturally be disposed to ask me to explain my apparent versatility, and to declare the therapeutic principles which guide me.

I stated at the beginning of this lecture, that it is a frequent occurrence for young girls on arriving in Paris to have suppression of the catamenia. Change of residence is enough in itself to produce this result, without there being any change in the manner of living. Young girls who have lived a number of years in a provincial boarding-school, on moving to a similar institution in Paris, where the *régime* is evidently the same, often experience an interruption of several months in their courses; and young girls removing from Paris to the country similarly suffer. We constantly have in our wards servant girls from the country, who, for some months after their arrival in Paris, have ceased to menstruate. Here, then, is amenorrhœa from a cause for the removal of which we are not called upon to do anything, unless unfavorable symptoms arise.

Some girls begin all at once to menstruate regularly: others, and they are probably the majority, menstruate very irregularly for two or even three years, without seeming to have the slightest derangement of health. This state is often a cause of anxiety to mothers: but the most prudent course to follow under such circumstances is to abstain from all interference, at least until some disturbance in the health render it necessary to institute decided treatment.

I need not tell you that in acute diseases, menstruation is only slightly modified (as my colleague M. Hérard has shown), but that, on the contrary, in chronic maladies, it first become irregular and then, in the majority of cases, ceases altogether.[1] We can understand why a very strong morbid antagonism should powerfully modify ovulation, a function only occasionally in exercise, when we see that it disturbs those functions which are more immediately necessary for the maintenance of life.

Suffering women too frequently attribute aggravations of their maladies to suppression of the menses. Though, sometimes, acerbations arise under the influence of an abortive menstrual congestion, yet the disturbance thereby induced is generally transient, and unimportant. There are cases, however, in which menstrual congestion may be regarded as the cause of severe symptoms in young girls of hemoptysic tendency. Whilst the economy is preparing for the great work of ovulation, which constitutes so important a part in a woman's life—as important in her as the work of reproduction is in every animal and vegetable organised species: it produces an excitation which pervades the whole system, and gives rise to fluxions and hemorrhages, the latter being particularly apt to occur in woman. Women, likewise, often have headache, swelling of the breasts, and hemorrhoidal congestions: epistaxis is also usual, and hemoptysis, in those of tubercular predisposition. There is greater reason to fear accidental, when there is a cessation of the normal hemorrhage.

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[1] HÉRARD:—Mémoire sur l'Influence des Maladies Aigues sur les Régles. [*Actes de la Société Médicale des Hôpitaux de Paris, fascic. 2me,* 1852.]

Although physiologists allege menstruation to be always connected with an act of generation—abortive or non-abortive—in this sense that it is always preceded by the development and rupture of an ovule—clinical observers must protest against so absolute a statement. Under the influence of some mental emotion, or some morbific cause, we often see the catamenia reappear some days after they had ceased; and in such cases, it is difficult to believe in the existence of a preparatory ovarian work. Sometimes, the blood appears a few minutes after the mental cause by which the woman has been affected, just as occurs in epistaxis. Although I do not dispute that there is a relationship between ovulation and hemorrhage from the uterus, I am disposed to consider the latter as a simple coincidence, determined especially by modifications of the uterine nervous system, similar to those produced by many other causes.

The study of the phenomena which precede each menstrual period has an important bearing on the treatment of amenorrhœa, for a reason on which I will afterwards enlarge, and which for the present I will merely mention. The reason is this:—once the catamenia have been suppressed, it becomes impossible to calculate their return from their usual and normal epochs. I now recur to the phenomena of which I was speaking.

Pretty generally, menstruation is preceded by a certain change of disposition, not always very appreciable by the physician, but recognised by those in habits of intimacy with the woman. Frequently, also, there is a feeling of general discomfort and loss of appetite. In a very considerable number of cases, one or several small pustules of acne appear on the lips and chin. These are the primary signs of a menstrual period, which are apart from the organs of generation. The other more important precursory signs specially belong to the reproductive organs: I refer to turgidity of the mammæ which become painful, and the lobules of which are more distinctly felt than at other times: also, to heat in the sacral region, sensation of weight in the loins, slight leucorrhœa, frequent desire to make water, and (in many who are generally constipated) to a tendency to diarrhœa. I need not add, Gentlemen, that in most chronic diseases, there is a slight exacerbation of the symptoms, which, in many cases, has a positive significance.

You will at once perceive, Gentlemen, the importance of questioning, with very great minuteness, women suffering from amenorrhœa, so as to elicit from them the presence or absence of the precursory signs which I have now succinctly noticed. It is necessary clearly to bear in mind that it is exceptional for hemorrhage from the uterus to occur at other times than the menstrual periods. It is vain to attempt to cause a return of the menses after they have ceased eight days—for such a purpose it would be useless to employ any imaginable diversity of measures; on the other hand, you would find it a matter of extreme difficulty to adopt any medication sufficiently bad to prevent their reappearance at nature's appointed term. If it be so difficult to induce the sanguineous discharge in absence of the normal preparative changes, what success can we hope for from giving any so called emenagogue to a woman suffering from amenorrhœa?

Opportunity is as important a consideration in pathogeny as in therapeutics. A man has just had an attack of gout—not his first attack: consequently, he is a gouty subject. If he have got quite over the attack, he may commit excesses at table or with women, or keep late hours, without immediately inducing a new paroxysm of gout. But when the gouty principle has remained pent up for a long time, when it is ready prepared, accumulated within the system, the slightest exciting cause will be sufficient to produce a violent attack. I may say the same in respect of herpes, megrim, asthma, and other diathesic diseases. A person when sweating profusely, may get drenched with icy rain, or he may ten times rest in a marsh exposed to cold and damp, without suffering on return home the least pain, or even without a cold in the head. Eight days later, however, a half-open window may give him inflammation of the lungs or acute rheumatism. This is explained by there being no predisposition in the first, and in predisposition existing in full plenitude in the second state. In the same manner, you can understand that the general preparation in the economy which terminates in menstruation increases, and accumulates from the cessation of one catamenial flow up to the commencement of another; and also, that the influence of medical treatment will be quite different according as the measures are employed to-day or three weeks hence.

Emenagogue treatment, Gentlemen, is a very complicated affair; and it may be said, that if there be a method of treatment deserving of the epithet *emenagogue*, there are very few medicines entitled to that appellation.

Do you suppose that you treat amenorrhœa in the same way in a strong plethoric woman, as in a subject of extreme anæmia?

Do you suppose that if the morbid antagonism of inflammation of a lung or of any other organ has caused suppression of the menstrual flux, you ought to proceed as you would in the case of a young woman whose courses have been abruptly stopped by putting the feet into cold water? Gentlemen, the mention of these well-known matters is sufficient to point out to you the difficulty of the treatment.

In women and in men, there are necessary functions in constant operation: as the cutaneous, renal, and hepatic secretions go on uninterruptedly, you can understand that sometimes the physician requires to do very little to excite these different secretions, the system being always in a state of readiness to perform them. There exists a continuous functional aptitude, which can be augmented by means very slightly active.

But menstruation is an intermittent function—transient, and, in a sense, accidental. So numerous are the circumstances which disturb it, that it is easy to see that therapeutic difficulties have to be encountered when it is wished to augment or regulate it. This function is not so essential to the adult woman that it must be performed: it is also easily disturbed when the general functional harmony is seriously deranged. When this general derangement exists, it is useless to attempt to restore the menstrual function by emenagogues. The primary indication is to restore the equilibrium; and then the special excitant of the uterus becomes an important weight in the balance. The menstrual function will be tardy in re-establishing itself, not only so long as an antagonistic flux continues, as is the case in certain acute and chronic phlegmasiæ, but also in the diasthesic diseases (such as chlorosis, albuminuria and diabetes), which exceedingly alter the constitution of the blood and disturb the harmony of the nervous system.

If in chlorosis, far example, the permanent and necessary functions, such as calorification, innervation, diuresis, and diaphoresis, are so strangely and inveterately perverted, what must it be in respect of an accidental function such as menstruation?

It is, therefore, Gentlemen, too evident to require further remark, that if the menstrual flux be prevented by the antagonism of a fever or a phlegmasia, the sole object of the physician must be to combat fever or phlegmasia. Should plethora be the cause of the evil, it is by diminishing the mass of blood, and reducing its plasticity, that the uterine flux will be facilitated; whereas, if the functional derangement be attributable to chlorosis, the proper treatment will consist in administering ferruginous preparations and tonics. These different means, so opposite in their nature to one another, are not emenagogues; and yet they are the most powerful agents in emenagogue treatment. As this statement may seem a little obscure and odd to some of you, let me explain it, so that you may quite understand my meaning.

The better the health, the greater the regularity with which all the functions are performed; and if the advent of disease accidentally excite serious disturbance of the functions, their restoration to a normal state will be brought about simply by restoring the general health. In fact, cessation of the causes of disturbance is sufficient to enable the system to return to its right working order. The treatment which effects the cure is not an excitant of the disturbed function: it simply restores order, and so enables the laws which preside over the economy to resume their empire unhindered by any obstacles. Thus it is that tartar emetic, digitalis, quinine, iron, bloodletting, and many other remedies apparently so opposite in their properties, prove themselves to be emenagogues, in the same way that they act as excitants of the secretion from the lungs, kidneys, and liver, in virtue of their power to restore the general health. They have no special properties. But, Gentlemen, the health being re-established, there may still remain some functional incertitude, some hesitation in the functions to resume their usual course; and it is in such circumstances, that it is useful to employ special excitants such as I am now going to consider.

There is no excitant emenagogue with which I am acquainted so efficacious as the general warm bath: to impress this fact on your minds, it is sufficient to remind you, that after a somewhat protracted hot bath, nearly every woman experiences uterine congestion, manifested by lumbar pains, a feeling of weight at the lower part of the abdomen, leucorrhœa, and an increase in the occasional flux, which often appears before the normal epoch. To obtain, however, the desired result, the bath must be given, at least, thrice a week, and repeated daily when the menstrual period is imminent. By-and-by, I will recapitulate the signs which I have already mentioned; and on which I cannot too much insist.

Bleeding from the arm, when the precursory menstrual signs exist, is a measure of immense potency; and it is not unusual for the uterine flux to appear an hour after the bloodletting. I need not tell you, Gentlemen, that this heroic treatment would scarcely be opportune in chlorotic women, or in those who have long been sufferers from chlorosis.

The application of leeches ranks next after bleeding from the arm; and as it causes less alarm to patients and their relations, it is a measure which, in general, is much more easily accepted. This, however, is a matter regarding which some explanations are required. It is very essential to know the number of leeches which ought to be employed, and the place to which they ought to be applied. First, I will speak of the place of application.

Some physicians think that the leeches ought to be applied to the labia majora, basing their opinion on I know not what theory, and also, perhaps, on the practical fact, that the application of a small number of leeches to any particular place, determines a great congestion to it. The proceeding is apt to occasion great inconveniences: it very often leads to local engorgements, furuncles, and boils. It has also, when the bites are healing, the serious drawback of exciting intense itching, which may sometimes occasion troublesome sensations in girls, leading them to contract bad habits. I now always apply the leeches to the inside of the knees; and I have never seen this practice prove less efficacious than the other. It possesses, moreover, the advantage of enabling the physician to arrest the bleeding, with the greatest ease, by making pressure on the condyles of the femur or the head of the tibia: and this is the more necessary that the application of leeches acts much less by subtracting blood, than by causing congestion. So convinced am I of this, that I invariably advise the bleeding to be stopped by agaric, as soon as the leech falls off; and I obtain equally good emenagogic results, the patient having only lost a few grammes of blood, a consideration of great importance. This practice is repeated on three successive days. Should the first application cause the menstrual flux to appear, I do not prescribe a second: should the flux stop, I order another application of leeches to be made.

It is quite true that the subtraction of even this small quantity of blood enfeebles chlorotic subjects; but it is a remarkable fact, that the reappearance of the flux in a natural manner, and with increased abundance, is often a precursor of a restoration to health. Notwithstanding the enfeebling which this treatment causes, I do not the less recommend it, because it really renders very great service.

When the stomach will bear it, the tincture of iodine ought to be administered three times a day, from five to fifteen drops being given each time in a weak infusion of saffron: this is a powerful emenagogue. It ought to be taken continuously for several weeks.

You have so often heard of iron as an emenagogue, that it is incumbent on me to make some remarks to you on that subject.

On chlorotic subjects who have amenorrhœa, iron seems to act as an emenagogue. On the other hand, when they have menorrhagia, iron acts as an hemostatic, which shows that this therapeutic agent is neither an absolute hemostatic, nor emenagogue, but a medicine which seems either to suppress or cause menstruation by re-establishing the health under normal conditions—conditions under which menstruation ought to take place.

By this very summary enumeration of the chief measures which ought to be adopted to restore menstruation, you may have observed that iron, the last which I have mentioned, is not absolutely an emenagogue; so little has it the properties of an emenagogue, that in a healthy woman, it rather diminishes than augments the monthly flux. It ought, consequently, to be considered as a *relative* emenagogue: the others are *absolute* emenagogues, as they will, in certain states of a woman's health, augment or cause the menstrual flux, provided there be not a too frequent repetition of the bloodletting of which I have spoken.

You recollect the remarks I made a little while ago on the necessity of considering *opportunity* in employing this or that remedy: never is this more absolutely necessary than in emenagogue treatment. Under ordinary circumstances, neither iodine, bloodletting, nor baths, will cause a return of the menstrual flux when it has ceased; but if the preparatory work has begun in the system, these measures will possess great emenagogue power. In amenorrhœa, from menstruation not having occurred a long time, there is no way of recognising the signs of the precursory work going on, and no way, consequently, of recognising the *opportunity* for employing emenagogues.

However, Gentlemen, by exercising vigilant attention, the physician may find out the exact time for prescribing emenagogues.

Before speaking of the signs by which to recognise the opportune time for action, let me remove a prejudice, which I regret often to see possessing a strong influence on the minds of physicians. I do not suppose that there is a single medical practitioner who believes in lunar influence, and is not perfectly aware that out of one hundred women, there is not one perhaps in whom the menstrual flux corresponds with the same phase of the moon. Many women maintain that they have their courses on particular days of the month, for many months in succession. However, on coming to exact count and reckoning with them, when they have been obliged to note the exact dates of a series of epochs, it is easy to satisfy oneself, and not difficult to convince the women themselves, that there is very little foundation for their assertion. This, Gentlemen, is a matter of some importance: in place of reckoning the epochs by the revolution of months, it is necessary, in each case, to compute the return of a period, by the time which, in the particular individual, has been ascertained to separate one period from another. This is an important matter for observation, when it is proposed to administer emenagogue remedies at the epochs when the catamenia are presumed to be due. But we must clearly bear in mind, that computation is impossible when there exists amenorrhœa of some months' duration. The suppression of the menses which occurs during pregnancy is purely physiological: it has, moreover, this curious feature, that during the first three or four months, most women have very distinct menstrual signs; and experienced accoucheurs are well aware that abortion generally takes place at a menstrual period: accoucheurs, also, from a dread of the fluxionary phenomena which thus appear, enjoin absolute rest at these periods upon women liable to miscarry. But when the amenorrhœa is not caused by pregnancy, the fluxion to the organs of generation at once loses its usual regularity, and no longer furnishes data whereby to calculate the probable return of the courses; and experience teaches us, that the sanguineous flux may reappear at very irregular and, indeed, at quite indeterminate intervals.

I required, Gentlemen, to enter into these details, to enable you to understand the utility—I would even say the necessity—of instituting treatment at one time, and avoiding to institute it at another. I have already told you, the approach of menstruation is announced to women by a certain amount of discomfort, by altered disposition, by swelling of the mammæ, by leucorrhœa, and by a frequent desire to pass urine. Both physician and patient ought to have their attention constantly directed to these phenomena, because it is at the time of their appearance, and only then, that the more direct excitants of the catamenia find their opportunity. Then it is, that the application of a few leeches, or such a bleeding from the arm as I have spoken of, brings on the menses. Then it is, that the tincture of iodine, saffron, and ammonia produce effects which are decidedly emenagogue. Then, also, it is that protracted baths will prove most efficacious.

When the signs of fluxion have passed, we must not continue the use of these means: we must wait for a new indication to employ them, and take special care to make use of them during the increase, and not during the decline of the fluxionary condition. When there is nothing in a woman's state to indicate congestion of the ovaries or uterus, the time is not so opportune for the physician's intervention. Then, it is necessary without any reference to the periods, as they are not recognisable, to proceed, with continuous medication, employing such remedies as iodine and iron, taking care, of course, that there exist no contra-indications to their being used.

Some physicians have recommended as a more direct measure for causing menstruation the establishing each month an artificial menstruation, a proceeding which has its advantages. In pursuing this plan, we must bear in mind the normal interval which, in each individual we have to treat, separates one period from another. For four or five successive days, a very prolonged hot bath ought to be given in the morning; and there ought to be introduced into the rectum, in the evening, a suppository containing five centigrammes of tartar emetic, or twenty-five centigrammes of rue or sabine. When a fluxionary movement has been excited by these measures, a leech ought to be placed to each knee for three days in succession, care being taken to stop the bleeding as soon as the leech falls off. On the following month, this treatment is repeated: but it is very necessary to bear in mind that nature does not easily respond to our injunctions, and that, frequently, the return of the fluxion which indicates coming menstruation will occur during the interval between the epochs which we have determined to be the menstrual epochs. When this occurs, we must follow the plan which I have just been pointing out.

LECTURE XCIII.

PELVIC HÆMATOCELE.

Physiological and Pathological Anatomy of Pelvic Hæmatocele.—Catamenial Hæmatocele: from Hemorrhage into the Fallopian Tube; Excess of Fluxion, or Deviation in the flow of the Sanguineous discharge, is frequent, slight, and often recurs.—Accidental Hæmatocele from Ovarian Hemorrhage, Alteration of the Parenchyma, or Varix of the organ is a rare and almost always a mortal malady.—Hæmatocele from Blood Ascending from the Uterus by the Fallopian Tube, and being Effused into the Peritoneum.—Cachectic Hæmatocele.—Hæmatocele caused by Alteration of Blood.—Tubal Hæmatocele.—Diagnosis: Tumor behind or around the Uterus.—Intra-peritoneal, Catamenial Hæmatocele.—Extreme Palor.—Slightness of Peritoneal Pain.—Intra-peritoneal, Accidental, or Ovarian Hæmatocele: slight Hemorrhage from Rupture of the Hæmatic Pouch: Acute Peritoneal Pain.—Extra-peritoneal Hæmatocele: slight Pain and slight Hemorrhage.—Differential Diagnosis: Phlegmon and Abscess of the Lateral Ligaments, Extra-uterine Pregnancy, Hydatid Cysts of the True Pelvis.—Treatment: Surgical Intervention to be avoided.

GENTLEMEN:—The case to which I wish to call your attention is that of a girl of sixteen, who has been admitted to Saint-Bernard's ward. She has only menstruated once; and that was two months and a half ago when resident in the country. She came to Paris, two months ago, in the capacity of domestic servant: she was then jaded, and had suffered from mental distress. On November 7th, the day of her admission to the hospital, she had the symptoms of continued fever. Two days later, there was no longer any doubt as to the nature of the febrile malady: the tongue was dry, the teeth were covered with sordes, and there was diarrhœa: the pink coloured lenticular spots were seen: there was pain in the abdomen, particularly in the right iliac fossa. The other symptoms were—a rapid pulse, drowsiness, low delirium, and mucous râles in the chest.

Towards the end of the second week, she had a better appearance, but it did not last long: she soon fell into a state of deep adynamia; and on Thursday 21 November, that is to say, about, probably, the eighteenth or nineteenth day of the disease, she died.

At the autopsy, we found an effusion of reddish serosity, free from clots, in the pelvis. In the midst of this effusion, was a tumor as large as a hen's egg. It belonged to the right ovary, and did not adhere to the neighbouring parts: there was no trace of peritoneum. The effused serosity weighed about from one hundred and fifty to two hundred grammes.

The right Fallopian tube had no adhesions: its fimbriated extremity was much injected: in the cavity of the tube, about its external third, there was a small quantity of sanious, grey-coloured, muco-purulent fluid. There was a tumor on the ovary resembling an egg in size and form: it was brown, smooth on the surface, and without false membranes. On its descending part, there was a projecting clot the size of a pea, which was hard, to a great extent fibrous, and was strangled by an ulceration across which it was continuous with another clot situated in the centre of the tumor. On making an opening in the posterior part of the tumor, it was ascertained that we had to do with a hæmatic cyst, which had contained a very large clot, which had partly escaped by the ulceration. Before opening the cyst, it was remarked, that the red serosity exuded drop by drop from the ulceration, which was imperfectly closed by the clot. The hæmatic cyst, therefore, was the source of the intra-peritoneal effusion, which most probably was effused during the last hours of life, as there was no trace of peritonitis.

The walls of the cyst were formed by the serous membrane of the ovary, lined internally by several very thin layers of fibrin, yellow, and easily detached. The cyst rested on the ovary, and was in direct communication with the cavity of a Graafian vesicle, the walls of which were velvety, and the seat of the original apoplexy. In the same ovary, other Graafian vesicles presented small apoplexies. It appears, then, that we surprised a retro-uterine hæmatocele in progress of formation, consequent upon rupture of a hæmatic cyst of the ovary.

Let me add, that there was no trace of ovarian pregnancy: I discovered no vestige of a fecundated ovum in the cyst, which only contained serosity, clots of blood, and fibrinous deposits. The uterus was perfectly healthy, and its neck was untorn. The hymen was intact.

I believe that the ulceration of the walls of the hæmatic cyst was dependent upon the state of the patient's general health.

The glandular patches of Peyer presented large, deep ulcerations, in some of which the reparative process was in progress.

In listening to the account of this autopsy, you must have been struck with the special anatomical lesion—the ovarian hæmatic cyst, with its ulcerated wall. I remarked that a sanguineous intra-peritoneal effusion was found; and this must have led you to form a correct view of those peculiar effusions, to which much attention has been directed during the last ten years, and to which has been given the name of *retro-uterine hæmatocele.*

Bear in mind this fact: but beware of supposing that it is the rule, and that every hæmatocele has its origin in a hæmatic cyst of the ovary.

I consider that there are two principal kinds of hæmatocele. One of them has its origin in the ovary, which I, therefore, call *ovarian* hæmatocele: this I look on as the least usual form, although it affords the most frequent opportunities of studying the pathological anatomy of the affection. The other may be called *tubal,* or better still, *catamenial,* a name which recalls the fact that the hæmatocele originated in a hæmorrhage, during the catamenial flux, into the mucous membrane of the tube or its pavilion. This is the most common form, and may be seen several times in the same patient. It is not so serious as ovarian hæmatocele; and as it seldom has a fatal termination, little is known regarding its pathological anatomy. This circumstance, likewise, explains why the existence of this kind of hæmatocele is less generally admitted.

I long believed that I was the first to describe catamenial hæmatocele: indeed I had been for a long time under the impression that it had never been mentioned by any one else when my lectures were published in the *Gazette des Hôpitaux* in 1858; but in 1860, a prior claim to the theory was made. I am far from complaining of having been preceded by a distinguished observer: the theory will not be looked on as less sound because I am not its sole defender: I regret, however, that the honorable clinician to whom I refer should have vindicated his title to priority with so little moderation. In any case, I am far from adopting an exclusive explanation of the manner in which the hemorrhage takes place in hæmatocele: this statement is sufficiently proved by my division of hæmatoceles into ovarian and catamenial.

The etiology of hæmatocele is dominated by a great function: that function is menstruation. In catamenial hæmatocele, the exaggeration or deviation of the function may be said to constitute the disease: the hæmatocele is only a symptom, and the hemorrhage is the necessary and immediate consequence of the modification of function. Increased determination of blood to the tube and its expanded extremity produces either sanguineous exudation or rupture of a vessel; and the catamenial blood is poured into the cavity of the peritoneum. The effusion is often considerable, and may give rise to the symptoms of internal hemorrhage. In ovarian hæmatocele, the ovary is diseased in the first instance, and rupture of the softened organ, or of a hæmatic cyst, may be only an accident which will be often found to have menstruation as its determining cause; and at other times, external violence, such as a strain, a fall, or the jolting of a carriage. These causes may likewise lead to the rupture of the utero-ovarian veins, if they be varicose.

Another kind of hæmatocele may result from reflux of the catamenial blood from the uterus towards the tube, and its subsequently passing into the peritoneal cavity. This will be observed in cases of excessive menorrhagia, or when there exists an accidental or congenital obstacle to the natural exit of the blood from the uterus into the vagina. Dr. Bernutz has ably supported this theory of hæmatocele, admitting, however, at the same time, that there are other forms of the affection.[1]

There is yet another form of hæmatocele which merits special mention, although in the cases upon which its description is founded, the hæmorrhage has often been incomplete, remaining in the tubes without passing into the peritoneum. The term *cachectic* is applicable to this form, because it is met with in cases of purpura, malignant jaundice, scarlatina, measles, and small-pox; that is to say, in circumstances in which the blood is modified in its constitution in such a way as to have a great tendency to exude by the mucous membranes. Intra-tubal sanguineous tumours, unconnected with previous fecundation, occurring during menstruation, or at a longer or shorter period after the menstrual epoch, testify to the facility with which the mucous membrane of the tubes may become the seat of hæmorrhage. In the cases of Barlow, Simpson,[1] Hélie (of Nantes), and Laboulbène, there was cachectic tubal hemorrhage which might have passed into the cavity of the peritoneum, as was observed to have taken place in the cases reported by Scanzoni[2] and Barlow.

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[1] BERNUTZ:—Clinique des Maladies des Femmes.

The etiological fact to which I wish specially to direct your attention is the determination of blood to the genital organs at the time of menstruation. This determination which presides over the reproductive function is not met with exclusively in the higher orders of organic life: it is met with in the lower classes of animals and in plants. The generative act is always accompanied by a very remarkable fluxion. In spring, the young shoots of plants are the seat of a special congestion. This afflux of sap at a determinate time has no other end than the development of the shoot destined to bear the flower. We also see an afflux of the juices towards the delicate organs of reproduction till the time has come for the pistil to receive the pollen secreted by the stamina. The flower is then full-blown: all its parts are rigid; but immediately after the great act of reproduction has been accomplished, as soon as the perpetuation of the species has been assured, the congestive afflux of sap disappears, and all the organs soon become withered.

In the lower animals, we also see congestion presiding at the fissiparous and gemmiparous processes.

In the human species, the principal functions cannot be performed without a considerable afflux of blood to the organs. Thus, during long continued intellectual occupation, there is a flow of blood to the head: during mastication and stomachal digestion, there occurs a profuse secretion of saliva and gastric juice, which can only be explained by congestive fluxion of the salivary glands and glands of the stomach.

During ovulation, this afflux is still more remarkable. In that act, congestion proceeds to hemorrhage: at each spontaneous ovulation, there occurs rupture of a Graafian vesicle, and each rupture is accompanied by a slight hemorrhage which becomes the origin of an ovarian hæmatic cyst, of the nature of those which have been so well studied by Charles Robin.[1] Generally, this hemorrhage is slight, the process of enucleation of the ovule is accomplished in a few days, and the ovuliferous vesicle is untorn except at one point which limits the source of the hemorrhage. We shall afterwards see, that if the reflux of blood to the part, or the softening of the ovary, occasion profuse hemorrhage, a hæmatocele will be formed. There is a sanguineous afflux towards the external organs of generation, as well as towards the ovary at the time of ovulation. The vagina and labia majora then acquire a temporary increase of vascularity and temperature. During the time of rut, the cow has a glairy, sanguineous discharge from the vulva: the mammæ become swollen, hard, and full of colostrum. All these phenomena result from the erethismal congestion of the organs of generation.

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[1] Simpson:—(James Y.) On Vesico-uterine, Vesico-intestinal, and Utero-intestinal Fistulæ, as results of Pelvic Abscess. [*Edinburgh Monthly Journal* for October, 1852.]

[2] Scanzoni:—Traité Pratique des Maladies des Organes Sexuels de la Femme. No. 304. Paris; 1858.

Similar changes take place in a woman. Each menstrual period is accompanied by a remarkable turgescence of the mucous membrane of the vagina and vulva. There are also pains in the breasts, and in the regions of the kidneys and ovaries; these pains being indicative of local congestion.

These statements rest on experiments and clinical observations; and are confirmed by pathological anatomy when death occurs at a menstrual period. Normal anatomy, if properly studied, will show that considerable congestion must take place during the exercise of the ovarian and uterine functions.

Professor Rouget's beautiful experiments have explained the way in which erethismal congestion takes place in the genital organs of women. This ingenious anatomist after immersing the dead body in a warm bath, injected the vessels, and thus demonstrated the erectile property of the plexuses which envelope the ovary and form its vascular bed. During the artificial erethism caused by injection of the vessels, every part of the generative organs become turgid. The ovary, the lateral movements of which are limited by its ligament and mesentery, was seen to bear itself upwards and towards the highly injected fimbriated extremity of the Fallopian tube, by which it was grasped and capped.[1] There was thus presented to the eye, a picture of the physiological occurrences which take place at a precise moment; and when we see a vascularity so intense, and so liable to very active congestion, we readily understand how the mucous membrane of the tube, which (according to M. Béraud) contains vascular rings, may become the seat of hemorrhage at the menstrual period. We can also understand, that under the same circumstances, the ovary may become softened, apoplectic, varicose, originating, possibly, a catamenial hæmatocele. But in the event of the latter supposition being realised, there must previously exist a structural change of the ovary; for a healthy ovary covered with its peritoneal coat could not, from the mere menstrual effort, become the source of any notable hemorrhage; otherwise, hæmatoceles would be infinitely more common than they are.

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[1] Robin (Charles):—Note sur Les Hémorrhagies des Vésicules Ovariennes. [*Mémoires de la Société de Biologie:* 2me série, T. III, p. 139 for 1856. Paris: 1857.]

Hemorrhage from the mucous membrane, on the other hand, may take place without these membranes having undergone any previous change: for its production, it is only necessary that there exist previously intense congestion, or an alteration in the constitution of the blood. Thus, normal physiological ovarian hemorrhage, not being sufficient in quantity to produce a hæmatocele, and abnormal ovarian hæmorrhage requiring for its occurrence a previous alteration of the parenchyma of the ovary, we have a right to suppose that ovarian hæmatocele is very rare. On the other hand, the tubal mucous membrane, from its special structure, from the hæmorrhagic tendency which it possesses in common with all mucous membranes, from the determination of blood which takes place as an accompaniment to menstruation, there arises a condition of all others the most favourable to hemorrhage, and so, likewise, to the formation of catamenial hæmatocele.

I am, I confess, not much disposed to believe that the peritoneum can be the source of a hemorrhage: sanguineous effusion seldom takes place from serous membranes which have not been previously the seat of inflammation, and then the inflammation is often of a special nature, as in cancerous and tuberculous peritonitis. The cases reported by Dr. Tardieu[2] seem to me to point to catamenial hæmatocele, that is to say, to tubal hemorrhage, particularly when it is remembered that the ovaries were carefully examined, and found to present no alteration of structure.

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[1] ROUGET:—Recherches sur Les Organes Erectiles de la Femme; sur Ovulation et la Menstruation. [*Journal de la Physiologie de l'homme et des animaux*, 1858.]

[2] TARDIEU:—Observations Pratiques de Médecine Légale sur les cas de

We have just seen that there are two principal causes which giv rise to hæmatocele—organic lesion of the ovary, and functional lesion of the Fallopian tube; on this subject, scientific opinion is divided. Some authors attribute hæmatocele exclusively to one or other of these causes; and others are eclectic, accepting both theories, and believing that under determinate circumstances ovary or tube may be the starting point of the bleeding.

Among the exclusives may be ranged Nélaton, Denonvilliers, Huguier, Lenoir, and Laugier, who believe that the seat of the hemorrhage is always in the healthy ovary, when it occurs at the time of ovulation; and in the diseased ovary, when it occurs at other times. Formerly, I used to maintain, that the tube always was the seat of the hemorrhage. New cases have modified my opinion, and I am now eclectic in my views: that is to say, I hold with Drs. Puech, A. Voisin, Bernutz, and Gallard, that there are various causes of hæmatocele. I consider that there are two principal causes—one ovarian and accidental with organic alteration of the ovary, and the other tubal and catamenial without organic alteration of the tube.

The existing dissidence of opinion is explained by the rarity with which hæmatocele occurs, and the still greater rarity of autopsies in such cases. Then again, the autopsies, being frequently made long after the commencement of the hemorrhages, are far from advantageous for the elucidation of the pathogenetic problem. The ovary, tube, and peritoneal cysts may produce such structural changes as sometimes to render it impossible to determine whether the hæmatocele is within or without the peritoneum: and *a fortiori*, it is often impossible to make out whether the organic lesions of the ovary are primary or secondary. In saying that catamenial hæmatocele does not cause death unless the amount of hemorrhage be excessive, which is very unusual, and that ovarian hæmatocele proves fatal by inducing peritonitis, which is more common, I mean, that autopsies having been chiefly performed under the latter conditions, many observers have been led to believe only in hæmatocele of

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Mort Naturelle et de Maladies Spontanées qui peuvent être Attribuées à un Empoisonnement. [*Annales d'Hygiène Publique et de Médecine Légale. Paris:* 1854. *T. II, p.* 157.]

ovarian origin, produced either by an ovarian cyst, an apoplexy of the ovary, or a partial suppuration of the ovary.

In catamenial hæmatocele, on the contrary, death occurs only in exceptional cases; and, consequently, the autopsies which would demonstrate the hemorrhage to have originated in the tube itself, or in its expanded extremity, must necessarily be also exceptional. However, the archives of science contain a certain number of necroscopic observations possessing a twofold interest, from their establishing, *first,* that there had been tubal hemorrhage which had passed into the peritoneal cavity, and *second,* that the patients had not sunk in consequence of a moderate hemorrhage into the peritoneum, but rather from a state of general disease, as in the cases related by Barlow, Simpson, Laboulbène, Proust, and Hélie. In these cases, there was no inflammation of the peritoneum, and the death of the patients was caused by purpura, small-pox, measles, and scarlatina.

In these cases, there is no ground for supposing, with Bernutz, that the hemorrhage had taken place by a reflux from the uterus through the tube into the peritoneum, seeing that both the superior and inferior orifices of the cervix were free, as was proved in Scanzoni's case by the catamenia being abundant, and in Laboulbène's case by the occurrence of an extensive flow of blood from the uterus at the commencement of the symptoms. It is, therefore, more reasonable to suppose that the mucous membrane of the tube may, like that of the uterus, be the seat of hemorrhage, and that that hemorrhage from the tube may be poured into the peritoneum.

It is the more rational to believe in tubal hemorrhage, as it has been shown by the anatomical researches of MM. Rouget, and Béraud, and by the admirable inquiries into the physiology of menstruation of Lee, Raciborski,[1] and Pouchet,[2] that there is a catamenial transudation from the Fallopian tubes.

These statements do not rest on mere theories. Follin and Oulmont have reported two cases in which they found, *post-mortem,* that the intra-peritoneal hemorrhage had proceeded from the mucous membrane of the oviduct. Three cases of Dr. Tardieu, reported by Dr. Auguste Voisin,[1] support, in my opinion, the doctrine of tubal hæmatocele. The cases are of so much importance that I will quote the passage in which they are described. It is to the following effect:—

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[1] RACIBORSKI:—De la Puberté et de l'Age Critique chez la Femme au point de vue physiologique, hygiénique, et médical, et de la Ponte Périodique chez la femme et chez les mammifères: Paris, 1843.—Du Rôle de la Menstruation dans la Pathologie et la Thérapeutique: Paris, 1856.—Traité de la Menstruation: Paris, 1868.

[2] POUCHET: Traité Positive de l'Ovulation Spontanée et de la Fécondation dans l'Espèce Humaine et les Mammifères. Paris, 1847.

"We have met with examples of this remarkable kind of effusion into the true pelvis, behind the uterus, occurring in two young women without any relation to conception or attempted abortion: death took place in both so rapidly, that suspicions of poisoning arose, and led to judicial inquiries, in which there was not elicited any other cause of death than that which I have stated. One of these women had been married three weeks; and the catastrophe according to the avowal of the husband might be attributable to excessive coitus. The other was a young Jewess whose malady began after sexual excesses with some students."

In addition to these particulars, which Dr. Tardieu communicated to Dr. A. Voisin, I must add, that in the case of a third woman (in which there was an autopsy) death followed a kick which the woman received on the left haunch from her husband.

Dr. Tardieu ascertained, by examination, that there was intra-peritoneal hemorrhage; and he thought that it was the result of a sanguineous exudation from the peritoneum. It was thoroughly established by Dr. Tardieu, that there was no lesion of the ovaries, Fallopian tubes, or pelvic blood-vessels. There was no apoplexy of the ovary, nor was there any rupture or ulceration of a hæmatic cyst. As it was not reasonable to suppose that there could be an extensive hemorrhage from the peritoneum, unless it had previously been the seat of inflammation, I was obliged to regard the sanguineous discharge as coming from the tubes. Now, Dr. Tardieu does not say that peritonitis had existed. On the other hand, it was proved that the first two patients were suffering from excessive coitus, in consequence of which the utero-ovarian blood-vessels were exceedingly congested. In the third case, the patient was subjected to external violence during menstruation: the intra-peritoneal sanguineous effusion was very extensive.

Thus it seems, that during or after excessive sexual indulgence, or after injuries received during menstruation, extensive intra-peritoneal hemorrhages may occur, without its being possible to trace them to any other source than the mucous membrane of the tubes; because, there being no lesion of the ovaries or utero-ovarian vessels, they are only to be explained by the structure of the mucous membrane of the tubes.

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[1] Voisin (Auguste):—De la Hæmatocèle Rétro-utérine, et des Epanchements Sanguins non enkystées de la cavité peritonéale du petit bassin. Paris: 1860.

In making this statement, I am not denying that hæmatocele is produced by reflux of blood from the uterus into the peritoneum, when there is an obstacle to the menstrual flow by the natural passages, such cases having been observed by Ruysch, de Haen, Delpech, and Bernutz.

There is reason to believe, however, that in women in whom there is absence or extreme narrowness of the vagina, the deep-seated sexual organs are slightly developed; were menstruation as abundant in them as in other women, the bad consequences of retention of the menstrual flux would be very common in the former; but this is not the case. Pretty frequently, we meet with cases of atresia of the vagina in women between twenty and twenty-five years of age. Several years ago, I attended, along with Professor Velpeau, a young lady twenty-seven years of age, who, for seven years, had had monthly attacks of dysmenorrhœa. On examination, we found that there was complete absence of the vagina; but by introducing the finger into the rectum, we discovered a tumour which, from its situation and form, was evidently the uterus. Every month, this individual experienced pains in the hypogastrium and loins: the breasts became hard: and at the period of spontaneous ovulation, she had severe abdominal pains, which were, I believe, dependent on the fall of an ovule, accompanied by some blood, into the cavity of the peritoneum. This young lady menstruated internally. A febrile condition continued for some days, the pains then diminished, and ceased in eight or ten days. Similar symptoms recurred every month; and at each of these recurrences, there was a beginning of catamenial hæmatocele.

I concur in the opinion of Drs. Richet and Devalz,[1] that in a varicose state of the broad ligament, rupture of vessels may occur, and become the source of a hæmatocele which will generally be extra-peritoneal, because the varicose veins are situated between the two folds of peritoneum covering that ligament. I also concur with

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[1] DEVALZ: Thèse. Paris, 1858.

Dr. Devalz in believing that the varicose condition of the large veins, by extending to the veins of the parenchyma of the ovary, may cause œdema, partial softening, and apoplexy of the ovary. There will take place something similar to that which we see in varicose ulcers of the lower extremities: hemorrhage (extra or intra-peritoneal) may be the consequence of the ovarian softening or ulceration. It is evident that hæmatocele originating in this way must be of rare occurrence.

The special varicose condition of the veins may predispose to apoplexy of the Graafian vesicles, and become the beginning of hæmatic cysts, which themselves constitute one of the immediate or accidental causes of hæmatocele.

I have just been mentioning the part which rupture of varicose veins may have in the formation of hæmatocele: Dr. Devalz relates the cases of Drs. Richet, Depaul, and Gueneau de Mussy, which show that hæmatoceles from rupture of the utero-ovarian veins have their seat in the peritoneal cavity, or in the sub-peritoneal cellular tissue.

Bear in mind, that whenever you wish to demonstrate venous hemorrhage on the dead body, you must, as Dr. Devalz recommends, previously inject the utero-ovarian veins. I regret that in a very remarkable case of hæmatocele, which occurred in the wards of Dr. Alfonse Becquerel, reported by Dr. Benjamin Ball, no such injection was made, for had it been made, the source of the hemorrhage might have been discovered. This inquiry possessed a twofold interest: the effusion was sub-peritoneal, and yet the right ovary was so changed, that it was nearly impossible not to consider it as the origin of the hemorrhage. Keeping in mind these two cases, it is impossible not to admit that rupture of the ovary in the ovarian mesentery may occur. However, in the history of the case, it is not stated that rupture of the ovary was ascertained to have taken place: perhaps, from some abnormal opening in one of the engorged tubes, the catamenial blood escaped into the substance of the broad ligament.

I cannot do better than read to you the details of this case, reported very carefully by Dr. Ball, who, when he made the necropsy was thoroughly well informed upon the subject now before us.

"An unmarried woman was admitted to Dr. Becquerel's wards on 18th January, 1858. She was carried on a stretcher to the hospital when in a state of profound coma.

"Two days previously, it was stated, this woman had been obliged to take to her bed in consequence of a chill during menstruation. The symptoms having rapidly become aggravated, it was considered necessary to have her taken to the hospital on the third day of her illness.

"At that time, her condition presented all the symptoms of the utmost gravity. The face was pale, and the features contracted: the forehead perspired profusely: the lips were white, the extremities cold, the pupils dilated: respiration was stertorous, and there was a little sanguinolent foam coming from the mouth: the skin was nearly insensible. The pulse, 140 in the minute, was thready, irregular, and very rapid. Respiration was so noisy that auscultation of the heart was impossible. Mayor's hammer was applied without obtaining any result. An hour later, the patient died.

"*Autopsy.* On opening the abdomen, no liquid flowed out, nor was any sanguineous effusion found in the peritoneum: there were neither false membranes, nor adhesions of the intestinal convolutions.

"After having raised up and detached the mass of small intestine, there was discovered a large sanguineous accumulation prominent on the sides of the uterus: that organ, pressed towards the pubes, had left its print upon the coagulated mass. When this mass was raised up, it was found that the effusion had taken place into the peri-uterine cellular tissue, between the uterus and rectum; the coagulum extended down to the vicinity of the anus, pushing forwards and upwards the posterior wall of the vagina: the tumor nearly filled the true pelvis, the cellular tissue of which was destroyed: the broad ligaments also contained coagulated blood. The peritoneal covering of the posterior surface of the uterus was partially detached by infiltration, which extended up to the middle of the body of the uterus, passed above the mass of coagulum, and was continuous with the peritoneal coat of the anterior surface of the rectum.

"The Fallopian tubes were shrouded in the tumor, and *contained soft, red clots.* Upon washing out the tubes, it was found that their mucous membrane was red, swollen, and vascular.

"The left ovary, completely infiltrated with blood, was converted into a blackish pulp, in which no remains of the structure of the organ could be detected. The right ovary presented no appreciable alteration of tissue.

The effused mass had the consistence of currant jelly, and a blackish red color. No membranous envelope encysted it, and it contained no fluid.

"The uterus may be described as of ordinary volume, although its exact dimensions were not measured. Its cavity contained no clots, but *its mucous lining presented a vascular arborescence.* The os tincæ was thickened: its lips were split in different directions: *the uterine orifice was half open.*

"The state of the other abdominal viscera was normal."

In 1855, when M. Nélaton's theory was generally accepted, M. Laugier, perceiving that it did not explain the majority of the cases of hæmatocele, set himself to demonstrate that, unless the ovary had previously undergone structural change, it could not originate an intra-peritoneal hæmatocele. He arrived at the following conclusions:—

"1. Spontaneous ovulation, as had been previously stated, is the immediate cause of retro-uterine hæmatocele.

"2. Physiological congestion of the ovary during spontaneous ovulation, with persistent aperture of the Graafian vesicle, does not cause hæmatocele.

"3. To produce hæmatocele, there must be a state of excessive congestion: such a state is sometimes induced by accidental causes during, or a few days after, menstruation. Abortion is not (as had been erroneously supposed) an immediate cause of hæmatocele.

"4. The chief immediate causes of hæmatocele are the returns of spontaneous ovulation, which gradually augment the volume of the hæmatocele.

"5. The successive ovarian vesicles open into the hæmatic cyst, and there remain; so that the ovary is destroyed by a small number of spontaneous ovulations, occurring under the conditions presented by the organ when the formation of an hæmatocele has commenced.

"6. As rupture of a Graafian vesicle affords passage for the blood from the ovary, the cyst of the hæmatocele is generally intra-peritoneal.

"7. A character which belongs in common to spontaneous ovulation and hæmatocele is unilateral abdominal pain, the seat of which is the ovary during vesicular evolution.

"8. Rut in animals may cause ovarian congestion followed by rupture of the ovary, that it is to say by pathological phenomena similar to those belonging to retro-uterine hæmatocele." [1]

M. Laugier directed his attention only to ovarian hæmatocele; and was ready to admit other etiological explanations if sufficiently demonstrated to him.

There now remains only one variety of hæmatocele for me to speak of—a variety which I believe to be exceedingly rare. Though I have not found in the cases related by Dr. Tardieu sufficient proof of tubal hæmatocele, I cannot absolutely deny that under certain special conditions, which cannot be very definitely described, exudation of blood into the peritoneal cavity may take place irrespective of any cancerous or tuberculous cachexia. Though I have seen no such case, and though I do not know of any, yet, reasoning from analogy, I am constrained to admit with Dr. Bernutz that these effusions may take place into the peritoneum, just as they take place into the cavities of the pleura, pericardium, and arachnoid; while, I repeat, however, that I believe such hemorrhages to be very rare, unless the serous membranes, or their contained organs, are the seat of cancerous or tuberculous deposits.

I have directed your attention to the varied etiology of retro-uterine hæmatocele; but I still require briefly to indicate the points in relation to the proximate causes of sanguineous effusion which are indicated in the remarks I have now made.

In cases of catamenial hæmatocele, the menstrual epoch is the exciting cause: possibly, sexual intercourse during menstruation may be a determining cause. In animals, however, coitus takes place only during the period of rut, the rut being a state similar to menstruation, and it is very unusual to meet with hæmatocele in female animals, who have not had connection with the male, yet you will find in Dr. Laugier's memoir a very curious case of sanguineous effusion into the peritoneum of a cow, which occurred during rut, though it is not stated that the animal was fecundated. The hemorrhage came from a hæmatic cyst of the ovary, from which there flowed several litres of blood. After menstruation and ovulation, external violence, jolting in a carriage, a fall, or a strain, may cause rupture of an ovarian cyst, of a tubal cyst, or of a varicose vein; and when we reflect upon the great development and turgescence of the ovarian vascular plexuses during pregnancy, the rarity of hæmatocele from rupture of the ovarian veins seems remarkable, till we remember the fact, noticed by Devalz, that, under the circumstances, the walls of the veins acquire great thickness.

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[1] LAUGIER:—Mémoire sur l'Origine et l'Accroissement de l'Hématocèle Rétro-utérine. [Presented to the Institute, 26th February, 1855.]

Hæmatocele can be detected by digital examination *per vaginam*, and by palpation. It is ascertained by digital examination, that the cervix uteri is situated immediately behind the pubes, that the vagina is shortened, that, behind the cervix there is a hard tumor, which has often been mistaken for the body of the uterus in a state of retroflexion, but which is recognised as being independent of the uterus from having neither the form nor consistence of that organ. This tumor is a hæmatocele.

By abdominal palpation, the bladder being empty, a hard round tumor is detected behind the pubes, which tumor is not the uterus, as can be proved by practising simultaneously abdominal palpation and digital examination of the uterus. It is then ascertained that the uterus is enclosed by, and rests upon, a more or less resistant and, sometimes depressible tumor, which occupies the whole of the lower and posterior part of the true pelvis. By introducing the index finger into the rectum, we can circumscribe the sanguineous effusion, so as to determine its lateral boundaries; whilst by abdominal palpation, we can ascertain its superior boundaries, so very extensive sometimes as to reach up even to the umbilicus.

It is unusual, however, for the tumor to be so large. It frequently extends more on one side than the other of the uterus, this inequality in development not always bearing any relation to the situation of the origin of the hemorrhage. Sometimes adhesions, resulting from previous inflammation of the uterus and its appendages, may explain the enclosure of the sanguineous effusion, and its greater bulb on one side than on the other.

The following are the symptoms of hæmatocele: during, or irrespective of, menstruation, a woman is all at once seized with acute pain in the abdomen, soon becomes pale, the skin in all parts of the body loses its color, and the slightest movement is unendurable: already, there is reason to suspect that the case is one of intraperitoneal hemorrhage: the pain and palor lead you to this conclusion, the correctness of which will soon be confirmed by physical examination. The degrees of intensity of these two symptoms—the pain and the palor—or the predominance of one of them, may give you a clue to the origin of the hæmatocele.

Should the pain be very acute, and the palor not very marked, while the pulse is very low, thready, and compressible, while vomiting and diarrhœa exist, you may conclude that the amount of hemorrhage is small, that its seat is the peritoneal cavity, and that very probably its source is the ovary. The amount of hemorrhage is small, because it is furnished by the rupture of an ovarian cyst or disorganised ovary: in the latter case, the effused blood may be changed, and of so irritant a character as to produce peritonitis.

On the other hand, the hemorrhage is catamenial and profuse, when the palor is extreme and the abdominal pain slight: the source of the hemorrhage is then the tube and its expanded extremity. The palor is extreme, because the mucous membrane of the tube has rapidly yielded a profuse hemorrhage: and the pain is not great, because the effused blood, not being altered, has produced very little irritation of the peritoneum. Experiments which I formerly made with Dr. Leblanc proved, that normal blood does not irritate serous membranes; but that blood which has become modified by being external to the vessels exceedingly irritates such membranes.

Let me add, that these two symptoms in conjunction—palor and pain—are particularly observed in cases in which the cavity of the peritoneum is the seat of the hæmatocele; so that, when they are less marked, the inference is that the hæmatocele is extra-peritoneal. To make a complete diagnosis, it is necessary to make a digital examination by the rectum and vagina, and to employ abdominal palpation.

The two affections with which retro-uterine and peri-uterine hæmatocele have been confounded are pelvic peritonitis and peri-uterine cellulitis. You know that after delivery, abortion, or cauterization of the cervix uteri, it is not unusual to have pelvic cellulitis and pelvic peritonitis. Besides mentioning that both these inflammations are almost exclusively observed under these circumstances, I ought to state:—1st, that in pelvic peritonitis, the sero-purulent effusion, which is its consequence, never gives rise to a *solid retro-uterine* tumor of as resistant a character as in hæmatocele: 2nd, that, on the contrary, in peri-uterine cellulitis, there is formed, in the first instance, a resistant tumefaction of the cellular tissue of the true pelvis and broad ligaments. The swelling is always greater on one side than on the other: the uterus generally inclines to the right or the left, and very seldom forwards: ultimately, after a variable period, fluctuation becomes evident, and the inflammation shows a great tendency to invade the iliac fossæ. Peri-uterine abscesses frequently open into the rectum, bladder, or vagina: and some of them point towards the inguinal region or crural canal: again, the inflammation may gain the anterior abdominal wall, and then the pus, detaching the peritoneum, makes an exit for itself above the groin.

When the hæmatocele is intra-peritoneal, it soon becomes encysted, and absorption of the serous fluid rapidly takes place, particularly when the hæmatocele is catamenial; and it is only in exceptional cases that the tumor evacuates itself into one of the natural reservoirs. It happens otherwise when the hæmatocele is of ovarian origin, the acute peritoneal irritation may then extend to the cellular tissue, and (as in extra-peritoneal hæmatocele) the tumor may open into the vagina, rectum, or bladder. When hæmatocele is extra-peritoneal, that is to say when it is situated in the cellular tissue of the true pelvis, the diagnosis may be made at the commencement of the hemorrhage; but at a later period, if there be inflammation of the pelvic cellular tissue, it is only the suddenness of the primary accidents which can give us a clue to the diagnosis.

I will not enlarge upon the diagnosis between hæmatocele and extra-uterine pregnancy: let it suffice to remind you, that Dr. Gallard, in his first memoir, was disposed to regard all retro-uterine and peri-uterine hæmatoceles as cases of extra-uterine pregnancy; but, besides the fact, that hæmatocele is met with in virgins, it is important to remark that the commencement of hæmatocele is sudden, while the beginning of extra-uterine pregnancy is latent, its progress slow, and the hæmorrhages to which it may give rise only observed at a period far removed from the period of impregnation.

I will not enlarge upon the differential signs of ovaritis and ovarian cyst. I would remark, however, that cysts of the true pelvis, situated in the sub-peritoneal cellular tissue, may, in women, be mistaken for hæmatocele. I refer to hydatid cysts which have become the seat of inflammation. The following case will show you the possibility of making an erroneous diagnosis:—

A young woman of nineteen, irregular in her menstruation, had experienced rather violent abdominal pains at the time of an imperfect menstrual flux. Some months later, she was suddenly seized with pain in the true pelvis, pain which particularly extended to the right side of the abdomen. Digital examination, *per vaginam,* enabled me to ascertain that the entire uterus was pushed towards the symphysis of the pelvis: there was a tumor as large as the fist situated laterally behind the uterus, and occupying the right side of the true pelvis: the rectum and tumor seemed to constitute one mass: digital examination by the rectum occasioned great pain. Pain interfered with defecation: the desire to make water had become very frequent. Absolute rest, low diet, and expectant medication were enjoined. Ten days after the commencement of the symptoms, there occurred anal tenesmus, and sanious discharge from the rectum, attended by comparative relief, and by collapse of the abdominal tumor. The stools, which had a dysenteric appearance, were examined daily with care: it was found that they contained no clots of blood; but, during three days, fragments of soft cystic false membrane. In a few days, the patient's health was completely re-established. I regret that the membranes thrown off were not examined by the microscope: my impression was, that they came from suppurating hydatid cysts of the true pelvis.

I at first thought that this young woman was the subject of hæmatocele, an opinion supported by the sudden commencement of the abdominal pain. It might have been supposed that there had been an extra-uterine pregnancy, the cyst of which had become the seat of inflammation. For months, this young woman had suffered from disturbance of the catamenial function. The expulsion of a false membrane, smooth and soft, resembling the envelope of an hydatid, and the simultaneous sudden disappearance of the tumor, led me to abandon the opinion, that there was either a sanguineous tumor or a fœtal cyst, and by exclusion, to conclude that the case was one of an inflamed hydatid cyst eliminated by ulceration through the rectum.

Cysts of this kind are met with comparatively seldom in the true pelvis, particularly during the first half of life, as appears from the researches of Charcot and Leudet. They do, however, sometimes occur; and when they become inflamed, they originate a group of symptoms which are of such a character as to demand notice in discussing the differential diagnosis of retro-uterine hæmatocele.

The details into which I have entered in reference to the formation of hæmatocele, and in reference to the seat and extent of the hemorrhage, point out that the prognosis cannot be the same in the different kinds of hæmatocele. Catamenial hæmatocele is not, in general, a serious affair: if there have not been much hemorrhage, the volume of the tumor soon diminishes, the pain disappears, and the uterus regains its normal position. If, on the contrary, the hemorrhage be profuse, the tumor has not only a much greater size, and involves a larger portion of the peritoneum, but induces an extreme degree of anæmia predisposing to new tubal hemorrhages, which, by continuing for some months, may augment the tumor, and prevent its absorption.

Last year, you may have seen in bed 8, Saint-Bernard's ward, a young woman with the symptoms of peri-uterine cellulitis: the tumor increased in size, became fluctuating, and yet showed no tendency to open spontaneously into any of the natural cavities. I resolved to make a puncture at its most salient point, that is to say, in the lower part of the abdomen, four or five centimeters from the linea alba. The progress of the phlegmonous abscess led me to believe, that adhesions had formed between the cyst and the walls of the abdomen; but I was greatly surprised to see from two hundred and fifty to three hundred grammes of sanguinolent serosity issue from the canula: the remainder of the tumor was composed of fibrinous clots. Immediately after the puncture, great relief was experienced by the patient, the fever ceased, and sleep returned. By slow degrees, the tumor was absorbed; and the patient left the hospital cured, after having exhibited, when under our observation, during more than a month, signs which led me to form an erroneous diagnosis. The principal causes of my mistake were the persistence of inflammatory symptoms, and the progressive increase of a fluctuating tumor. It must be noted that, for the first sixteen days, this woman had had bloody discharge from the vagina; and probably in this particular case, the tube had at the same time, poured into the peritoneum a constantly increasing quantity of blood: this patient had a devious, intra-peritoneal menstruation. It is well to remember that these hæmatoceles, which may be called (so to speak) continuous or intermittent, are often associated with a special condition of the blood predisposing to hemorrhages.

The remarks which I have made on the progress of extra-peritoneal hæmatoceles obviates the necessity of my discussing at any length the gravity of their prognosis. A considerable number of published cases sufficiently establish the frequency of inflammation of the subperitoneal cellular tissue. These hæmatoceles have a tendancy to open into the rectum or vagina; and as, even after these spontaneous openings, general poisoning has been observed, we ought to avoid opening them with the knife, unless there are special and urgent indications for the adoption of that proceeding.

There are, however, at the commencement of every hæmatocele, two indications demanding the physician's intervention, viz. hemorrhage and peritonitis. Should the hemorrhage threaten to continue, we must resort to local and general hemostatics: the application of ice to the abdomen, the administration of rhatany, sulphuric acid, and ergot of rye may be prescribed with advantage. If, on the other hand, the peritoneal pain be the predominating symptom, it must, in the first place, be calmed by narcotic, obtunding drugs: opium or belladonna must be administered internally, and applied externally by frictions to the abdomen.

When the hæmatocele is catamenial, that is to say, when it is most probably tubal, the principal object to be kept in view is to restrain the hemorrhage and prevent its recurrence.

It is often necessary to seek for the cause of the hemorrhage in the general state of the patient. If she have chlorosis, or anæmia, employ invigorating treatment, selecting in each case the particular medicine best suited to the state of the individual. Some patients, when placed under favorable hygienical conditions will lose all symptoms of chlorosis and anæmia: the open air, exercise, good food, and mental satisfaction will cure them. Others will require ferruginous preparations and bitters. Bretonneau recommended the preparations of cinchona in hemorrhages, and administered them successfully in the epistaxis which is so common in young people. I believe that, to prevent the renewal of hemorrhagic attacks seemingly associated with chlorosis or anæmia, we may, in cases of hæmatocele, advantageously employ the preparations of iron and cinchona.

Is it neceasary to discuss at length the value of puncture in the treatment of hæmatocele? The surgeons who recommend the tumor to be punctured by the rectum or vagina were the first to point out the necessity of abstaining from all surgical intervention, so long as there was no threatening of the cyst bursting into the peritoneum. Their caution is worthy of imitation; and indeed, I cannot too strongly advise you to follow their example. From numerous cases, it has been learned, that there is danger in establishing a communication between the interior of the cyst and the external air, particularly when the opening is made through the walls of a natural passage, the rectum or vagina, in which fluids undergo very rapid alteration. When there is an unquestionable indication of the necessity of surgical intervention, a puncture ought to be made through the anterior abdominal wall, provided it be united to the hæmatic tumor by adhesions. Proceeding in this manner, we avoid one cause of general poisoning, by avoiding doing anything to permit the contents of the rectum or vagina to enter the interior of the cyst.

LECTURE XCIV.

PUERPERAL PURULENT INFECTION.

Puerperal Fever is not a simple morbid state.—The Physiological State called "Puerperal."—It predisposes Lying-in Women and New-born Infants to a variety of affections, such as Peritonitis, Phlebitis, and Lymphangitis.—In these Puerperal Affections, there is a great Tendency to Suppuration.—A Primary Purulent Diathesis exists in Puerperal Women.—A Secondary Purulent Diathesis may exist, the consequence of Phlebitis, Inflammation of the Lymphatics, or the direct Absorption of Pus from the Placental Wound.—Secondary Purulent Infection of Lying-in Women and of New-born Infants is identical with the Purulent Infection consequent upon Amputations.

Gentlemen:—United under the generic denomination of *puerperal fever*, there are a great many morbid conditions which all in common possess pyogenesis as a characteristic. Is there such a thing, correctly speaking, as a puerperal fever? The several great efforts which have been made to describe this morbid entity have only served to show the great extent and variety of the pathology of the recently delivered woman.

In an academic discussion, which is still of recent date, the most competent observers expressed entirely different opinions as to the malady called puerperal fever; which shows that it is not *one* like such fevers as measles, scarlatina, and small-pox.[1]

Some regard puerperal fever as a local disease—a peritonitis or a phlebitis. Others look upon it as a general disease, like typhus, in which the lesions are only secondary, and are infinitely various. There are others again, who, while they admit the affection to be general, consider it to be the result of a purulent or putrid infection of local origin, such as phlebitis, or gangrene of the uterus. Then there is another class—perhaps the most sagacious—who attribute the diversity of opinion among observers to the diversity in the disease they describe.

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[1] De la Fièvre Puerpérale, de sa Nature, et de son Traitement: Communications à l'Académie Impériale de Médecine, par MM. Guérard, Depaul, Beau, Hervez de Chégoin, P. Dubois, Trousseau, Bouillaud, Cruveilhier, Piorry, Cazeaux, Danyau, Velpeau, Jules Guérin, &c. précédés de l'indication bibliographique des principaux écrits publiés sur la fièvre puerpérale. 8vo, pp. 464: Paris, 1858.

My own opinion is that in a matter of this kind, we must be eclectic. I believe that puerperal fever, like typhus fever, is happily a rare disease; and I also believe, that the different puerperal symptoms described as peritonitis and phlebitis are very common. Before describing the different forms which are assumed by puerperal symptoms, let us recall the physiological conditions of the pregnant and recently delivered woman. They constitute a special morbid predisposition—*a real diathesis.*

From the very moment when conception takes place, great changes occur throughout the woman's whole organism. The countenance acquires a peculiar aspect: generally, the features are drawn, the eyes are surrounded by a dark areola, the nose is pinched, and sometimes the face is covered with ephelides. The nipple, the linea alba, and the mucous membrane of the genital organs acquire a brown color: the follicles of the vagina, the labia majora, and mammary areola become increased in size. Menstruation is stopped: the breasts soon become swollen, and prepare themselves for the secretion of milk. There is sometimes increased salivary secretion, capricious appetite, vomiting, diarrhœa or constipation. The liver enlarges, and its icini becomes loaded with fat: the thyroid gland may show increased development, and even the heart may undergo well marked hypertrophy. The blood is modified in respect of the proportion of its component parts: there is an increase in the fibrin, and a diminution in the red globules: a soft blowing sound is heard in the vessels of the neck and at the base of the heart. There then exists a variety of chlorosis which was first described—and very well described—by Cazeaux.[1] Albuminuria is sometimes met with.

Such are the principal organic and functional modifications of pregnancy. They already constitute the puerperal state—a physiological state which may lead to disease, and is often manifested by a special—a *purulent*—pathological condition.

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[1] CAZEAUX:—De la Chlorose des Femmes Enceintes. [*Bulletins de l'Académie de Médecine. Paris,* 19 *Fév.,* 1850*: T. XV., p.* 448.]

Lorain[1] and Tarnier[2] have lately laid much stress on the fellowship of the morbid state of the mother and her new-born infant, and still more of the mother and fœtus. The infant, whether an appendage of the placenta or the breast, lives by its mother's blood. It must, therefore, share the conditions predisposing to disease which act on the mother, and must similarly receive at a given moment—at the time of an epidemic, for example—the same morbid influence. As the infant grows older, it proportionately loses this morbid solidarity. In the womb, the fœtus lives solely by its mother: but when surrounded by the atmosphere, on the contrary, it may, *proprio motu*, contend against its inherited maternal morbid conditions. It will, therefore, be less and less under puerperal conditions, the longer the interval of time since its birth.

We see then, that the fœtus shares, and the infant retains, for a period of varying duration, the maternal puerperal state. Both derive from the mother a puerperal state, and all its consequences, the chief of which is a predisposition to purulence.

Some have wished so to extend the puerperal condition in women as to include the condition at each menstrual period; and certainly, each menstrual flux is accompanied by an exfoliation from the internal surface of the uterus. In certain cases, the uterine mucous membrane exfoliates in shreds or in one entire piece; and when this sort of delivery is painful and difficult, morbid phenomena are observed similar to the ordinary phenomena of parturition: there occur renal pains, uterine colics, hemorrhage during and after the detachment of the mucous membrane, followed by a sanio-purulent discharge, and consecutive separation: this incomplete delivery, which accompanies the menstrual ovulation, lasts for some days, after which nature resumes its natural order, till next menstruation. Whilst these changes are proceeding, a modification shows itself in the performance of the functions, and in the temper of the individual. There is an afflux of blood to the ovaries and tubes, and almost always a sympathetic turgescence of the mammæ. All these phenomena, which are principally localised in the uterus and its appendages, are analogous to those which appear at the end of pregnancy and after delivery. I must remark, however, that the general state of the menstruating woman is different from the general state of the recently delivered woman: though in the former, the blood, the humors, and the organs have undergone modifications, they are not of so profound a nature as in the latter. The general state is different; and that is sufficient to account for the morbid predisposition to puerperal symptoms being less. There is, however, in the analogy of the states, matter of high clinical importance; and the cases of peritonitis and purulent infection observed among menstruating midwives during puerperal epidemics, strongly support the views of MM. Lorain and Tarnier.

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[1] LORAIN: De la Fièvre Puerpérale chez la Femme, le Fœtus, et le Nouveau-né: Paris, 1855.

[2] TARNIER:—De la Fièvre Puerpérale observée à l'Hospice de la Maternité. Paris, 1858.

The new-born infant, as I have said, has received, during intra-uterine life a special influx from the mother, which imparts to it a predisposition to her morbid affections. The umbilical wound, which corresponds with the placental wound of the uterus, may occasion symptoms analogous to those observed in suppurative uterine phlebitis, so that we may have identically the same purulent infection in mother and infant. The general conditions, therefore, of the new-born infant are nearly the same as those of its mother. There exists in both a physiological solidarity, showing itself in an excess of formative power [*puissance organisatrice*]: there is also in both a pathological solidarity, showing itself in a tendency to purulence, and in a remarkable similarity in the local affections [*localisations morbides*].

If it be correct to say that the puerperal state begins with menstruation, or, according to some pathologists, begins with fertilised menstruation [*menstruation fécondée*], and ends with lactation, it is unquestionable, that the puerperal symptoms most frequently occur, and are most severe, at the epoch when the puerperal state is most marked, that is to say, during the three or four weeks immediately following delivery, when the uterine wound exists. This puerperal state is of shorter duration in the infant, and generally terminates with cicatrisation of the umbilicus.

Both in the recently delivered woman, and in the new-born infant, there exists a general state of the system which creates a *morbid predisposition*, and also a local condition which may give rise to different morbid manifestations, all shewing in common one characteristic —the tendency to purulence: peritonitis, cellulitis, and metritis are affections of this kind. Should the purulent tendency invade the venous system of the placenta or umbilicus, the local condition may become the starting point of a general purulent infection of the system, analogous to purulent infection in one who has undergone an amputation.

Phlebitis and purulent infection are not, nevertheless, in my opinion, all that is necessary to constitute puerperal fever, any more than puerperal fever is peritonitis, as Gasc, Pinel, and Beau suppose, or angioleucitis as is believed by Cruveilhier, Nonat, and Botrel. There does exist an epidemic puerperal typhus; but in every case, it must have a wound as its determining cause.

It is necessary, therefore, to establish clinical distinctions. To maintain that all puerperal symptoms result from a fever, or are produced by an inflammation having various localisations, would be to contradict observation.

I hold, consequently, that there are puerperal symptoms, and that there is a puerperal fever properly so called. To-day, I do not intend to speak to you about puerperal fever, as there is no case of it in Saint-Bernard's ward: I wish to direct your attention to the purulent infection of recently delivered women, an infection of frequent occurrence; and with several cases of which I meet every year. The young woman whose case I am now going to recapitulate died of that malady.

She was delivered at the Maternity on the 29th November. The labor was natural. On the third day after delivery, she had slight milk fever; but there was no loss of appetite. On the tenth day after delivery, she left the hospital, walking home. Five days after returning to her lodgings, this young woman was seized with shivering, which recurred on the following days: on the 15th December, she was admitted to the Hôtel Dieu. Immediately after her admission to the hospital, she had a return of the rigors. She nevertheless said that she was not ill, and had no pain in any part of her body. Her pulse was rapid, small, and compressible. She asked for food; and had neither vomiting nor purging. Still, from several returns of the rigors taking place, I suspected purulent infection.

No pain was excited by palpation of the hypogastric region, or by digital examination *per vaginam*. This absence of pain was abnormal in a woman only fifteen days after delivery. The cervix uteri was soft: the mouth was open, and from it there issued a small quantity of fœtid sanious discharge. The uterus was movable. When the patient lay on her right side, it was impossible to detect by digital examination any tumefaction of the broad ligament of that side: as the introduction of the finger occasioned no pain, I neglected to explore carefully the left broad ligament. The prognosis in respect of the fever was very unfavorable from the frequent recurrence of the shivering, and from the patient having no feeling of being ill.

During the night between the 16th and 17th December, she again had shivering. At the morning visit on the 17th, there was a change of expression, and pain in the right shoulder: as the arm could be moved about, I concluded that there was no affection of the shoulder-joint, and that the pain was probably dependent upon the presence of a purulent deposit round the articulation. Next day, the 18th, there was a renewal of the shivering: pains were felt in the left shoulder, but in no other part of the body. There existed rapidity of pulse, profuse sweating, and redness over the cheek bones. The mental faculties remained unimpaired: there was neither squinting nor deafness. Respiration was anxious: râles were heard in both lungs, particularly in the lower part of the right lung: there was neither bellows-sound nor egophany. Vomiting, diarrhœa, and abdominal stains were all absent. Towards evening, respiration became embarrassed, the râles became coarser and more general, the power to expectorate ceased; and death occurred during the night.

At the autopsy, it was found that the uterus was larger and more flabby than it was fifteen or twenty days after delivery: there, was, however, nothing abnormal in its parietes. Its internal surface was smooth, and, except in the situation where the placenta had been inserted there was an absence of rugosity: at that point, however, there was no suppurating wound, the corresponding uterine sinuses were contracted on themselves, obliterated by small fibrinous clots: there were no traces in their interior of inflammation or suppuration. The neck of the uterus was bluish and soft, and its lips were jagged: on making a section of the cervix, no inflammation of its circular vein was disclosed. On making a double longitudinal incision through the walls of the uterus where the sinuses and utero-ovarian veins meet, no purulent collection was met with.

On the left wall of the vagina, in the part nearest to the cervix uteri, there were ten pustules, as large as the full-sized pustules of small-pox: a creamy pus flowed from them when incised. In the cellular tissue lining the vagina, in the part corresponding to the pustules, numerous small abscesses were observed, the presence of which gave the tissue the appearance of a sort of purulent sponge. The left hypogastric vein was carefully dissected: creamy pus, free, unmixed with blood, was found in its interior: there was no obliterating clot, and no phlebitis, although from the point at which the intra-venous pus was met with, the dissection was carried up as high as the common iliac vein. The pus, which could only come from the parts adjoining the vagina, or from the vagina itself, had been carried into the hypogastric vein, and subsequently drawn into the circulation by the other affluents of the same vein. This case seems to me to be one of the best for the demonstration of the direct passage of pus into the general circulation. This was the visible source of the purulent infection, of which the lungs, liver, spleen, and joints, furnished the irrefragible proofs.

The lung presented numerous ecchymotic spots which gave great value to the existence of small abscesses surrounded by ecchymoses and minute apoplexies. At the base of the lung, at its edges, there were abscesses the size of a haricot, of which some were and others were not fluctuating. The liver and spleen only contained superficial ecchymoses and yellow spots.

Within the articular cavities of both shoulder joints, there was a large quantity of pus. No appearances of purulent peritonitis or purulent pleurisy were visible.

Most assuredly, Gentlemen, you have been struck with the resemblance—the identity, I may say—between the case now described and cases which come under your notice in the surgical wards. Have you not recognised in that case the same purulent infection seen in persons who have undergone amputation? Observation during life and the autopsy, must, I think, have quite satisfied your minds on this point. The symptoms of purulent infection in a delivered woman, as in a person who has had a limb amputated, are frequent rigors, recurring several times a day, pains in different parts of the body, cerebral disturbance, a general state testifying the presence of a general disease, recognizable superficial abscesses, in the joints or subcutaneous cellular tissue, extreme prostration, and speedy death. Metastatic abscesses are found disseminated in the principal viscera, and purulent effusion in several of the synovial cavities. Finally, to complete the description of the resemblance between recently delivered and recently amputated patients, let me state, that in both, there is an opening for the entrance of the poison by the venous system.

In a surgical wound, a placental wound, a tear of the cervix uteri, a contusion of the vagina, a phlegmon, a phlebitis, we have the source of the general infection of the system—the origin of the pus, which, carried into the circulatory torrent, acts either as a foreign body or as a ferment, in such a way as to determine a morbid state of the organism, which almost always terminates in death.

Purulent infection is the same affection in the recently delivered and recently amputated patient. Why, then, does not the placental wound and the surgical wound always give rise to purulent infection?

For a long time, Gentlemen, it seemed as if the general affection known under the names of "purulent infection," "purulent absorption," "pyæmia," &c. belonged exclusively to the domain of surgery, because physicians had fewer opportunities of observing it, and because we are indebted to surgeons for the majority of works on phlebitis.

At a date not yet very distant, M. Tessier maintained, with great ability, the theory of puerperal fever in opposition to that of phlebitis.

At a later period, experiments on animals were made by MM. Darcet (de Castelnau) and Ducrest, which were afterwards repeated by Sedillot. The questions of purulent absorption, phlebitis, and lymphangitis were first supported and then assailed: those who will take the trouble to read the principal works on this subject will find that, looking at Tessier's doctrine from a clinical point of view, it is at least a close approximation to the truth.

This is a question in general pathology which I propose to discuss with you. It is one which every physician ought to study thoroughly; and I hope that our attentive examination of the predisposing and determining causes of purulent infection, will lead us to understand its progress and etiology, and enable us also to seize the indications of a preventive treatment.

Every disease, at the first, has two elements: there is the cause, properly so called; and the state of the economy recipient of the morbid impression. These two elements are always present—the first to follow out its results; and the second, to react upon, to combat the cause. The action of the cause will be principally either in respect of its quantity or in respect of its quality. The reaction of the economy will differ in each individual according to his varying aptitude for resistance.

Even specific causes sometimes encounter refractory economies, which rebel against their morbific powers. This refractory aptitude may be either natural or acquired. It is natural, when we see a number of individuals remain exempt during an epidemic: it is acquired, when, in virtue of a previous attack of the same disease, or in virtue of an antidote, the morbific cause is obliged to remain inoperative. A person who has once had small-pox will rarely have it a second time; and a person who has once been vaccinated is always protected for a longer or shorter period from a second attack.

In some persons, habitual contact with a disease confers relative or absolute immunity from it. Physicians, nursing-sisters, and hospital servants seldom take the eruptive fevers, to the contagion of which they are constantly exposed.

If a man, day-by-day, for a certain period, take a particular poison, he will at last be able to take large quantities of it without experiencing the slightest discomfort. In Hungary, as you know, arsenic is very largely used as an invigorating medicine; and, as I have often told you, certain wretched victims of epileptiform neuralgia of the face have acquired so great a tolerance of the preparations of opium that one has been able to take a litre of laudanum in a day, and another from thirty to forty centigrammes of the extract of opium in the twenty-four hours.

Opium and arsenic though toxic agents possessing a determinate, almost specific action, require sometimes to be administered in augmented doses to obtain the desired and usual effect. So it is with specific morbid causes. Though, as a general rule, a specific element acts with certainty and produces anticipated results, yet sometimes specificity claims its quota. Thus, for example, a particular individual who resists contagion when the malady is endemic is unable to do so when a great epidemic is prevailing, because, under the latter condition, the specific cause is more powerful or is more prolonged in its action. This remark applies to epidemics of cholera and small-pox.

The economy will resist an ordinary or specific morbid cause with a power varying according to the individual and the cause: the resistance will vary infinitely according to the varying receptivity of individuals and the varying receptivity of the same person at different times.

These, Gentlemen, are principles in general pathology, without which you will be stopped at every step in practical medicine. They enable you to understand how the effects of morbid causes vary according to the idiosyncrasies of individuals, the nature of the prevailing epidemic, or the medical constitution of the season.

The principles which I have now recalled to your recollection admit of being applied to purulent infection in amputated and recently delivered patients? Without these principles, how can we understand the extreme frequency of purulent infection at a determinate epoch? How can we without them, understand its gravity being relative to the nature of surrounding circumstances? Or without them, how can we explain its mildness under certain conditions which do not admit of doubt as to its being actually present, such as articular and numerous subcutaneous abscesses? Are you not thus led at once to perceive the importance of this relative mildness and gravity? Every surgeon and physician knows that there are cases of purulent infection which terminate in return to health. Yet, how often has it been written that purulent infection is inevitably mortal? We shall see by-and-by whether the etiology, the progress of the infection, and the morbid epiphenomena supply us with indications whereupon to deduce rules of prognosis and treatment.

There is undoubtenly a purulent diethesis, that is to say, a predisposition in virtue of which certain individuals form pus with very great facility. In some subjects, every wound undergoes protracted suppuration: there are others, again, in whom every solution of continuity cicatrises rapidly. In the former, there is a persistent predisposition to form pus; and in the latter, a natural tendency to secrete that plastic lymph by which the lips of wounds become agglutinated, and their surfaces covered with a cicatricial coat. In the two classes of cases, therefore, there manifestly exists a diversity of predisposition, the particular predisposition being usually allied to a particular state of the general system which it is impossible to define, as those under its influence may present all the conditions of apparent health.

At other times, this general state which constitutes the purulent predisposition, is consequent upon external violence: and at other times, again, it follows an eruptive continued fever from which there has been imperfect convalescence. So great is the alteration in the crasis of the blood after small-pox, that we may see the body covered with a numerous succession of sub-cutaneous and submucous abscesses. In these cases, the purulent diathesis seems to confine its manifestations to the subdermic cellular tissue: sometimes, however, in cases consequent on typhoid fever, we find, at the autopsy, numerous parenchymatous abscesses. You recollect the case of the young man under the care of Dr. Horteloup, who succumbed to advancing general purulent infection, which probably originated in ulcerated Peyerian glands, and the existence of which infection was proved by numerous pulmonary and intra-muscular abscesses. A prick of the finger when dissecting, sometimes also, though not often, gives rise to purulent infection: more frequently, however, as you know, it gives rise to a phlegmonous or special general putrid infection, which may almost always be recognised by the stools, the odour of which recalls the primary source of the infection.

The disposition to form pus which follows continued fever is not generally a serious element in the prognosis, except, of course, in those exceptional cases in which numerous abscesses form in the muscles and parenchyma of viscera. Perhaps, the cause of the exceptional gravity of purulent infection occurring as a sequel of fevers, consists in some special modification of the wounds which serve accidentally as the origin of the infection, whether it be in a suppurative mesenteric phlebitis, or in an ulcerative phlebitis permitting the direct passage of pus into the circulatory torrent while the benignity of this affection may be due in other cases to *the digestion of the pus.* How are we to understand why it is that in small-pox, when the dermis is in contact with so great a quantity of purulent matter, there should be no purulent infection unless we admit that the purulent matter becomes modified in some peculiar manner by which its noxious properties are removed? There is good reason for supposing that, in each pustule, prior to desiccation, a modification takes place in the purulent fluid by which it is deprived of its infectious power.

Whatever there may be in these explanations, it must be admitted that there is a class of cases, in which, notwithstanding the profusion of pus which bathes the surface of the dermis or the deep-seated tissues of organs, purulent infection is a very exceptional occurrence; while in another category of cases, the affection originates in a wound of small extent, or in an erysipelas. Let me remark that, in most of the cases which belong to this category, there has been observed a noxious epidemic influence, sometimes manifesting itself in unhealthy suppurations, erysipelas, phlebitis, causing great mortality among lying-in women and new-born infants.

While, however, we attribute its own share to the prevailing epidemic influence, we must admit the existence of individual tendencies, for many amputated patients and many recently delivered women placed under the same general influence escape: those who resist that influence are in the most favorable circumstances for organic resistance. We must, therefore, always take into account the general etiological influence, and the individual's power of resisting it. Though we cannot bring treatment to bear upon the epidemic cause itself, let us see whether it be not possible to place individuals in such conditions as will enable them to resist the epidemic influence.

Principal Theories of Purulent Infection.—1. Absorption of unaltered pus by the absorbent vessels.—The pus-globule inadmissible: only the serum of the pus is admissible. The Vascular Oscula of Van Swieten and Transverse Sections of Veins becoming Absorbing Mouths.—2. Purulent Fever of Haen and Tessier.—Pyogenic Fever of Lying-in Women of Voillemier.—3. Suppurative Phlebitis causing Purulent Infection of Dance, Velpeau, Blandin and Marechal.—Capillary Phlebitis of Ribes.—Pus in the Thoracic Duct.—4. Absorption of the Serum of the Pus.—Experiments of Darcet, of MM. Castelnau and Ducrest, and of Sedillot.

Before I state my views of purulent infection, it will not be uninteresting to recapitulate the principal theories which have been brought forward.

The theory of absorption consists in the belief that the pus of an abscess or of a superficial wound may, as pus, be absorbed by the absorbent vessels, and be carried by them into the torrent of the circulation: it is supposed that the pus which is thus transported by the blood to different parts of the body, originates metastatic abscesses. Boerhaave and Van Swieten are of opinion that this absorption takes place by the ossicula of the vessels, or by the transverse sections of the veins. The pus mixes with the blood, vitiates it (*inquinat sanguinem*), and so becomes the source of cacoplastic deposits in the viscera, the functions of which become implicated to such an extent as to indicate the presence of the most formidable maladies. Boerhaave and his commentator believe that the pus is absorbed *en nature*. As you are aware, this theory has been most severely criticised. The researches of anatomists hardly allow us to believe that pus can pass directly through ossicula, or absorbing mouths, which have no existence! But the entrance of pus in consequence of the lesion of vessels is not the less possible, as Boerhaave observes, *after amputations and operations for aneurisms*, or after extensive and deep wounds—*ingens vulnus factum*. Hunter, in his chapter on "ulcerative phlebitis," defends the opinion of Boerhaave: and every day, we see how easily pus enters the uterine sinuses without there having been obliterating phlebitis.

Van Swieten had the idea that the pus did not merely act as a foreign body in causing the numerous abscesses; but that its presence in the blood produced a *special fermentation* engendering pus in other parts of the body(1).

Morgagni, when speaking of wounds of the head, and Quesnay,[2] declare in favor of the absorption of pus *en nature* by the veins; but they add, that the pus being carried to, and arrested in, the liver, lungs, and other organs, becomes the cause of a secondary suppuration—an opinion adopted by Professor Cruveilhier,[3] based on experiments tending to show that a globule of pus, like a globule of mercury, acts as an excitant of inflammation of the lungs and liver to which it has been transported.

De Haen, the pupil of Van Swieten, but not his follower in this matter, believed in the existence of a purulent fever, *analogous to the fever of small-pox*. He thought that, in these conditions, the blood, in virtue of a special predisposition, contains the principles of purulence, which show themselves everywhere throughout the organism, after the manner of the virus of small-pox. He adds, that the cause originating the buffy coat on the blood is sufficient likewise to produce pus. Around these two opposite theories are grouped all the other theories. One party believes, that the organism is infected by pus absorbed at a certain point: and others teach, that it is the

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[1] Van Swieten:—Commentaries on the Aphorisms of Boerhaave.

[2] Quesnay:—Traité de la Suppuration.

[3] Cruveilhier:—Anatomie Pathologique, du Corps Humain: livraisons iv, vii, xi, xiii: folio, Paris: 1833. See also his article on Phlebitis in the Dictionnaire de Médecine et de Chirurgie Pratiques, T. XII: Paris: 1834.

organism itself which generates the poison, and spontaneously produces pus everywhere.

Tessier, who has defended the doctrine of purulent fever with remarkable ability[1] is satisfied to remark, that in amputated patients and recently delivered women, there is a great tendency to suppuration, a tendency which shows itself, he says, in three quite distinct forms, viz. in the purulent phlegmasiæ, the purulent state, and the purulent fever. He looks upon the purulent fever as a febrile state, under the influence of which active suppurations suddenly appear in various parts of the body.

The object of Tessier's work was to prove that there was no such thing as purulent absorption, and that phlebitis, lymphangitis, and every local phlegmasia, arose from the general purulent state. He considered, that it exists before every fluxion and every local phlegmasia—that the predisposition, the purulent diathesis, originates in the traumatic condition, the puerperal condition, or other serious morbid states of the economy; but that these conditions are only predisposing causes, physical pain and crowding being the determining, and perhaps not the least important, causes.

Gentlemen, I cannot adopt this too exclusive view of purulent fever maintained by M. Tessier, because I am now going to lay before you cases in which it is evident that the suppurative phlebitis, the lymphangitis, and the suppuration of the capillary vessels, were the source of the purulent infection; that is to say, of the infection of the blood. Still, I have pleasure in recognising the very happy manner in which M. Tessier discusses the question of the purulent diathesis.

Side by side with Tessier's purulent fever, we must place Voillemier's pyogenic fever of lying-in women.[1]

The doctrine of phlebitis has more scientific precision than that of purulent absorption; but resembles it in this respect, that it admits the infection of the organism to be consequent upon the introduction of pus into the blood. Dance, in 1828, ably maintained this theory, which, notwithstanding the powerful opposition of Dupuytren, was generally accepted.

The starting point, according to this theory, is suppurative inflammation of a vein: the infection of the blood is looked upon as secondary, and the metastatic abscesses, so to speak, as tertiary symptoms. This phlebitis, already described by Hunter, was anew investigated; and pathological anatomy soon showed that the bodies of patients dying with symptoms of purulent fever presented phlebitis of the stump, phlebitis of the capillaries, or phlebitis of the uterus. The cause of the phlebitis was discovered: it was discovered in the parenchyma of organs, in the osseous tissue, the diplöe of the cranial bones, and in other situations.

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[1] TESSIER: De la Diathèse Purulente. [L'Expérience: 1838.]

[2] VOILLEMIER: Histoire de la Fièvre Puerpérale, Pyogénique, observée en 1838, à l'Hôpital des Cliniques.—Reprinted in 1862 in the "Clinique Chirurgicale."

But is not this phlebitis, the source of the infection, itself really a primary manifestation of a special predisposition, without which the vein could not become the seat of inflammation? Without this predisposition, suppurative inflammation would then be the necessary consequence of every lesion of a vein: but this is not the case—suppurative phlebitis is, relatively, a rare complication.

Whenever a vein is involved in an operation, one of two things must occur:—Either, the secretion of plastic lymph, the wound healing by the first intention; or, cicatrisation by the second intention, after a necessary preliminary stage of inflammation. In the first case, the lips of the vein in the wound are glued to one another; and this condition is maintained by fibrinous plastic lymph secreted by the internal surface of the vein: this is adhesive inflammation. In the second case, the vessel remains open: a cruoric clot, adherent to the internal surface, projects from the opening, and mingles with the secretions from the surface of the solution of continuity. This clot has a free external extremity, and an intra-vascular extremity adherent to the serous coat of the vessel. The length of the clot varies with its distance from the point at which the collateral venous circulation is established in the same vessel.

What occurs in the second class of wounds takes place in respect of the placental surface of the uterus, which may, at every point, be compared to a bleeding wound. In both, obliteration of veins is accomplished by coagulation of blood. In the uterine sinuses, the obliterating clots seldom measure more than from one to two centimeters, which is explained by the rich venous circulation of the uterus for some days after delivery. But should venous inflammation occur, and spread by degrees, it is usual to find clots prolonged into the utero-ovarian and hypogastric veins, as is sometimes observed in the brachial and femoral veins, after amputations of the arm and thigh.

The secondary clots undergo very remarkable transformations; but it is not known whether these clots have the power of inducing purulent infection. My opinion is that purulent infection generally originates in inflammation of the vein nearest to the solution of continuity, that is to say, in the wound itself. This, in fact, is the situation in which the existence of pus has been demonstrated; and if there be no fibrinous plug, there is a direct passage for the pus into the blood. This fact admits of easy demonstration in the uterine sinuses and hypogastric and ovarian veins of women who have died within three weeks after delivery. Again, in these circumstances, it is not unusual to see in the placental wound several large open sinuses with thickened walls, canals of communication by which is conveyed the pus of the placental surface, and the pus, free or mixed with blood, which is found in the hypogastric and ovarian veins. In these cases, there is observed in the veins nearest to the source of pus, a sanguineous fluid identical in color and consistence with that of leucocythæmia. These facts were observed by Hunter, Clarke, and Hodgson, and are mentioned in their respective works: they may be easily confirmed by any one who will carefully examine the lateral parietes of the uterus in a recently delivered woman, and the veins nearest to the surface of the wound in a case of amputation.

Can the elongated clots in the veins be the source of purulent infection? In favour of an affirmative answer is the fact, that these clots, in softening, often present a purulent appearance; but I am doubtful of this being the source of the infection, because we can only detect by the microscope, even in the midst of this purulent appearance, granules of fibrin, no pus-globules being visible.

Suppurative phlebitis properly so called, which is observed chiefly in the large veins near wounds, may also exist in the capillary veins, as has been demonstrated in cases of ostitis and caries. Ribes observed capillary phlebitis in cases of erysipelas and gangrene.

We have now seen that pus deposited on the surface of a wound, or contained within a vein, passes directly into the torrent of the circulation, and so originates purulent infection. Are there any other sources of this infection?

During the last few years, there has been a belief that there can be no purulent infection without suppurative phlebitis, the purulent inflammation of the vein being the necessary cause of the infection:

without that inflammation, it has been held that there can be no passage of pus into the blood. After delivery, as I have already remarked, pus may be met with in the uterine veins, although these veins are not inflamed, not even at the point where pus is seen. It must, therefore, be admitted, that in these cases, the pus has been transported to the place where it is found. Again, I lately showed you a beautiful example of this transport of pus in the hypogastric vein, occurring in a case of purulent infection following delivery. It may thus happen, that one of the afferent branches of the hypogastric vein may be the seat of suppurative inflammation. I believe that it so happens in the majority of cases. For this reason, I wish to recall to your recollection two somewhat interesting cases, which prove that pus may exist in these veins, and may nevertheless escape the notice of conscientious attentive observers.

Lenoir, surgeon to the Hôpital Necker, had performed ablation of a sarcocele. Complete cicatrisation seemed to have taken place, when the patient was seized with repeated attacks of shivering; and died, having metastatic abscesses, which were seen on dissection after death. Lenoir himself dissected the veins which he supposed to be in a state of inflammation; and, after two hours of fruitless search, was unable to recognise any signs of phlebitis. Dr. Gubler, at that time a pupil in the wards, continued the dissection, and discovered inflammation of the prostatic venous plexus. The pus contained in the plexus freely discharged itself into the hypogastric vein.

In Marjolin's service, at the Hôpital Beaujon, a man was bled from the arm: he soon had pain in the arm, and probable phlebitis. Symptoms of purulent infection set in; and the patient died. MM. Castelnau and Ducrest carefully dissected the veins, in the situations in which they expected to find phlebitis: for four hours, their search was fruitless. M. Castlenau then anew examined the veins of the arm, and found pus in an inflamed vein opening directly into the brachial vein, in which there was an obliterating clot, and a free passage for the pus into the subclavian vein.

In both of these cases, the existence of phlebitis would have been denied, had not renewed search been made; and, certainly, no renewed search would have been instituted, had not a firm conviction existed in the minds of the observers, that pus would be discovered, to show the source of the general purulent infection. In similar circumstances, therefore, we cannot make our examinations with too much minuteness.

How many patients die after catheterism with symptoms of purulent infection! In the autopsy of such cases, never omit to examine the prostatic plexus, and the whole venous system of the pelvis, for there, the source of the infection will probably be found. You ought to know where to look. Recollect, that these are the chosen spots where pus is present in uterine phlebitis, as well as in phlebitis following operations on the testicle, urethra, bladder, and prostate.

It does not follow, that phlebitis is the sole source of purulent infection. In cases of purulent infection observed by Velpeau, Nélaton, and Denonvilliers, numerous metastatic abscesses were found on dissection after death, and yet, no pus could be found in the veins near the seat of the operation.

For example, amputation was performed in the case of a man admitted to the wards of Dr. Denonvilliers for comminuted fracture of the humerus. He died fifteen days after the operation, with all the symptoms of purulent infection. At the autopsy, enormous abscesses were found in the liver. All the superficial and deep veins of the upper extremity, from the surface of the stump to the right auricle of the heart, were very carefully dissected, without the smallest trace of pus being discovered. The remaining portion of the humerus was sawn in several directions; but not the minutest drop of pus was found in the spongy tissue of the bone.

Though disposed to admit the great value of cases such as I have now described, I am not without doubts as to their negative importance. It is possible, that the primary source of the infection may have existed in some situation which was not thought of. The sinuses of the dura mater may be the seat of purulent deposits consequent upon a seemingly unimportant attack of otorrhœa: again, in the course of a vein, there may be suppuration of a gland, cellular tissue, or parenchyma; and, from ulceration taking place, pus may enter the vein, causing general infection, although the lesion of the vein may remain undiscovered.

At other times, the source of the infection has been in the thoracic duct or in the receptaculum chyli. How did the pus get there? That matters little: there it was; and thence it might easily pass into the subclavian vein, and so into the venous circulation. In these cases, there was purulent infection.

You see then, Gentlemen, that suppurative phlebitis plays an important part in purulent infection. Pus may, however, exist in the veins without any trace of inflammation of the walls of the veins having been found: at other times, we neither find pus in the veins, nor phlebitis: but in such cases, the thoracic duct and lymphatic vessels have not always been examined; and as we have just seen, these affluents of the venous system may contain pus in cases of purulent infection. It consequently follows, that in the great majority of cases, purulent infection simultaneously exists along with pus in the venous system and its affluents. Adulteration of the venous blood by pus appears then to be the proximate cause of purulent infection: this theory has on its side an imposing majority of facts.

Nevertheless, the partizans of purulent fever see in them only coincidences; or rather, they regard metastatic abscesses and phlebitis as consequences of a general purulent state. From their point of view, there is no relation of cause and effect between the surgical or placental wound and the metastatic abscess, both being looked upon as results of the same cause, and as similar in their nature though different in their seat. The objection to this interpretation of the facts is, that purulent infection never exists without a wound, however small that wound may be. On the other hand, to this objection a counter-objection may be urged, viz. that there are many wounds without purulent infection.

Here, let me remind you, that certain conditions are necessary for the production of *suppurative phlebitis:* with most of these conditions, we are unacquainted. But this we know—that every wound of a vein is a grave accident, liable to become the source of the greatest danger. Surgeons have observed, that the knife is more apt to produce suppurative phlebitis than the actual or potential cautery; and that almost never is there anything to fear from venous lesions produced in crushing or tearing operations. We also know, that intra-venous suppurations, which predispose to purulent infection, are induced by comminuted fractures and articular wounds, by any active interference with the uterus during labour or at the time of delivery, by partial retention of the placenta, or by extensive lacerations of the cervix uteri. I may add, that individual and hygienical conditions, crowding, and the pain of an operation, by their action on hæmatosis and the nervous system, create a predisposition to purulence.—All this I accept within certain limits; but as you know, cases are recorded in which purulent infection declared itself almost suddenly in persons whose general state was satisfactory:

some were suffering only from otorrhœa,[1] varix, corns, or chilblains. Spontaneously, or consequent upon intervention with the knife or ligature, a vein having been touched, suppurates, and the patient dies with symptoms of purulent infection. Persons in good health, with the exception of some insignificant malady, are operated on in the most favorable conditions—a vein becomes inflamed, and the patients die: metastatic abscesses form: pus is found in the petrous portion of the temporal bone, in a vein at the point where it was ligatured, or only above the ligature: now I ask, are we, with all these facts before us, not to see the relation of cause and effect existing between purulent infection, between the general state, and the wound? That would be entirely to abandon logic.

Nevertheless, doubt was permissible: it was necessary to have recourse to experiments. At a period when attention was keenly directed to alteration of the fluids caused by the entrance of putrid matters into the circulation, Gaspard in 1823, and I, in conjunction with M. Dupuy of Alford, in 1826, came to the conclusion, that the injection of putrid matters into the veins of animals (dogs, horses, and sheep), produced symptoms similar to those caused by purulent infection, leading to death in a few days when the quantity injected was sufficient. On examination after death, the blood was found exceedingly altered, and numerous ecchymoses were observed in the parenchyma of the viscera and the sub-serous cellular tissue. After injecting from thirty to forty-five grammes of putrid pus into the veins of animals, we found tumors presenting a carbuncular appearance, and abscesses, which we called tuberculous abscesses [*abcès tuberculeux*]. I merely, at present, mention the results of the experiments performed by M. Dupuy and me conjointly, remarking, that when we made them, we had no intention of producing a state of poisoning similar to purulent infection.

MM. Renault and Bouley, in 1840, injected two centilitres of pus into the jugular vein of an apparently healthy mare; and, after the lapse of a few days, were not a little surprised to see the symptoms of acute glanders: it is worthy of notice, that by simply inoculating the matter of the nasal ulcerations of that animal, acute glanders was produced in another animal. I do not enter into any discussion regarding this experiment: I am satisfied to state the fact.

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[1] An interesting case of this kind is described at p. 231 of volume second.

M. Darcet in 1842, and MM. Castelnau and Ducrest in 1843, repeated these experiments upon dogs and rabbits. In 1844 and 1845, M. Sedillot made similar experiments with pus derived from different sources.

All these experiments show, that most animals into whose veins pus, varying in quantity and quality, has been injected, have presented, from the very first, prostration, a dejected appearance, and great restlessness: they have nearly all refused to eat, and have been exceedingly thirsty; some have had paralysis of the sphincter of the anus. Before the reaction which indicated a return to health, they have had profuse alvine discharges. When, by repeating the injections, the animals were subjected to successive and progressive poisoning, they succumbed in from five to ten days after the commencement of the experiment: on dissection, ecchymoses were found, particularly on the surface of the lungs, and numerous abscesses, of different dates, in the same organs.

It appears then, Gentlemen, that whether we inject pus into the veins of an animal, or whether it enter a vein as the result of inflammation in the case of a recently amputated or recently delivered patient, the symptoms and the lesions are identically the same. Purulent infection is produced artificially in the first case; and is the result of phlebitis in the second case. What more is necessary to prove, that in both cases the presence of pus in a vein is the cause of the purulent infection? This double check, clinical and experimental, is almost as conclusive as the experiments made to elucidate vaccination, the inoculation of small-pox, of syphilis, and of glanders.[1]

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[1] For the history of this question, and for all the experimental and clinical researches, see:—Darcet: Thèse Inaugurale, Mai, 1842:—Castelnau and Ducrest: Rescherches sur les Abcès Multiples comparés sous leurs différents raplorts; Paris, 1846. Sedillot:—De l'Infection Purulente ou Pyohémie; Paris, 1849. This work, rich in experiments and clinical observations, is the most complete treatise on this subject.—Dumontpallier:—Thèse Inaugurale; Paris, 1857.—See also, the works of Boerhaave, Van Swieten, De Haen, Stoll; and the more recent publications of Blandin, Velpeau, Cruveilhier, and Tessier.

Doctrinal Statement.—Parallel between Experimental Purulent Infection, and Clinical Purulent Infection.—Similarity of Symptoms and Anatomical Lesions.—Similarity of the tendency to Critical Evacuations by the Skin and Intestines.—Possibility of Recovery from Purulent Infection: Complex Etiology of Purulent Infection from Inflammation of the Large, and Capillary Veins: from Absorption of Pus itself: from Absorption of Purulent Serum, Assimilated, or Poisonous Serum.—Epidemic Purulent Fever.—Theory of Ferments applied to Purulent Infection: Experiments of Pasteur, Chalvet, and Reveil.—Treatment of Purulent Infection: to avoid the Causes of Phlebitis: there is no Specific: Endeavour to excite Crises, and to support the Strength.

Be on your guard, when successive attacks of rigors occur in a newly delivered woman, or in a patient who has a wound in any part of the body: for then, there is reason to fear that a certain quantity of pus has entered the blood, and that pyæmia is threatened. The shivering is soon succeeded by fever, sometimes a copious sweat covers the whole body, or a profuse diarrhœa sets in, after which all seems to return to its normal state, and the idea arises that an unnecessary amount of alarm has been created. Wait twenty-four or forty-eight hours! Rigors will then set in anew, sometimes accompanied by chattering of the teeth and horripilation; and these symptoms will perhaps recur several times within a few hours. The general discomfort increases, the patient has a presentiment of approaching death, reaction takes place slowly, and the pulse is rapid and weak. At other times, were it not for these strange shiverings, the patients would make no complaint; and yet, they suddenly experience pain in some particular part of the body, such as the calf of the leg, the knee, or a joint. The swelling and redness which accompany the pain disappear in a few hours; and then other parts of the body are invaded. Under such circumstances, you need not hesitate to affirm, that purulent infection exists, that is to say, that pus has entered the blood, and that there are abscesses in the parenchyma of the viscera, in addition to those visible on the limbs and trunk: nearly always, several metastatic abscesses are found in the lungs and liver.

Anatomical experience enables us to state that these abscesses exist, when by mere clinical investigation we could hardly even suspect their presence: it is not unusual for such abscesses to exist in organs although the functions of the organs do not seem to be disturbed. The liver or lungs may contain abscesses in different stages of evolution; and yet, pain in the hepatic region and chest, cough and expectoration, may all be absent.

Metastatic abscesses at the base of the brain may be sometimes diagnosed by the occurrence of strabismus, by an inequality in the dilatation of the two pupils, and by the patients complaining of diplopia and obscurity of vision. Delirium and coma do not show themselves till the last stage: at the commencement of the purulent infection, they afford no assistance to the diagnosis, but at that early period, important cerebral disturbance may be observed. There is a discord between the quiet state of the patient, and the gravity of his condition: he cannot understand why he is so carefully and attentively interrogated: he will tell you that he is not ill. This quiet state is a prognostic of very great danger. You remember the young woman who occupied bed 20, Saint-Bernard's ward, who told us that she was not unwell: and in respect of whom, nevertheless, I had no hesitation in affirming, on learning that she had had repeated rigors, and ascertaining the date at which she had first been attacked by them, that the blood was poisoned by pus, and that the source of the infection was in the genital organs, although I had not been able to recognise decided pain in these parts.

It is generally after the fourth or fifth day from delivery, that the symptoms of purulent infection declare themselves, because three or four days are required for the pus to be secreted in sufficient quantity from the surface of a wound or within an inflamed vein. When the symptoms do not appear till the fifth day, the delay has probably been caused by the obliterating clots having in the first instance prevented the pus from entering the blood.

It is easy to understand, that when four days have elapsed after delivery, the date of commencement may vary very much; for once pus has been secreted at any point in the venous system, it may enter the torrent of the circulation by the obliterating clot being broken or displaced by any sudden movement or blow. Persistent inflammatory action may cause softening of the clots, without there being a sufficient contraction of the vessel upon itself: then the pus secreted on the other side of the broken or softened clot may be poured intermittingly or continuously into the circulation.

There is a remarkable similarity between the principal symptoms of purulent infection and the phenomena observed in the experiments of Darcet, Castelnau, Ducrest, and Sedillot.

Each rigor announces the entrance of pus into the blood: for a time, variable in duration, the patient, or the animal experimented upon, struggles to eliminate the morbid poison from the circulation: profuse sweating, diarrhœa, and vomiting supervene: but as a new supply of the infecting fluid is always pouring in from the source, the patient becomes exhausted in useless efforts, and the great functions of the economy become almost simultaneously compromised: the stomach rejects food and medicine: there is constant diarrhœa: the circulation and respiration become accelerated: the mental faculties gradually fail: the face does not bear the 'impress of suffering: by-and-by, the breathing becomes jerking and quicker: the pulsations of the heart are more rapid: the body is covered with a cold sweat: at last, the patient dies without a struggle. The calm which characterises the last scene is perhaps explained by the blood being poisoned to such an extent as to carry death simultaneously to every organ, so rendering it impossible for any of them to maintain the struggle. Every part of the body, even the parts which seem to be perfectly healthy, are impregnated with the morbid principle, which seems, as Bérard remarks, to manifest itself by the repugnant smell which issues from every part of the body a few minutes after death.

Death is almost always the prognosis in purulent infection. It is obvious, that if a large quantity of pus be introduced into the economy, and carried to every part of the organism, it must speedily produce changes incompatible with life. On the other hand, it is easy to see that a small quantity of pus will not be sufficient to infect the whole of the blood: the infected portion may then be eliminated by the stools, by sweating, or by the renal secretion, in which case, the symptoms of infection will cease, provided there be no more of the infecting cause admitted.

This arrest may occur even when metastatic abscesses have formed: and after a time, these abscesses may themselves be absorbed without any new metastatic manifestation.

You recollect the young woman, aged twenty-eight, who after delivery at the Hospice des Cliniques, was admitted to our Saint-Bernard's ward, on the twelfth day after delivery, with all the symptoms of purulent infection, viz. recurrent shiverings, diarrhœa, vomiting, a sub-icteric tinge of the skin, and metastatic abscesses under the skin as well as in the sterno-clavicular and metacarpo-phalangeal articulations. All at once, the symptoms were arrested: the patient's general state improved: the fever, vomiting, and diarrhœa ceased: there was no recurrence of shivering: the appetite returned, the sub-icteric tint disappeared, and we saw that, little by little, *digestion* of the superficial, intra-articular, and subcutaneous metastatic abscesses was being accomplished. This *digestion* took place exceedingly slowly. Nothing interrupted the progressive return to health.

In this patient, in whom there was uterine phlebitis and entrance of pus into the blood, it is probable that the continuance of the passage of pus into the blood was prevented, either by a change occurring in the relation of the affected parts, or by obliterating phlebitis taking place beyond the seat of suppuration: as the infection was arrested, and its cause ceased, whilst the struggle was still possible, the patient triumphed over the first morbid effects. I was, therefore, justified, Gentlemen, in reminding you, at the beginning of these lectures, that in the practice of medicine, we must take into account the quantity as well as the quality of morbid causes, and likewise individual aptitudes for resistance. The case which I have now detailed strengthens this precept in general pathology.

Let the case of this young woman remain impressed on your memories, to make you less absolute in your prognosis, and to prevent you from losing all hope in these cases.

Recovery, then, is possible in a case of purulent infection. The experiments on animals led us to this belief: MM. Castelnau and Ducrest have shown, that animals into whose veins insufficient quantities of pus were injected returned to health after suffering for two or three days from fever and diarrhœa, whilst those into whose veins a succession of injections were thrown died with the anatomical lesions of purulent infection.

With such facts before the mind, it is natural to believe that death is not the inevitable issue in every case of purulent infection. What are the conditions required to modify the prognosis of an inevitably fatal issue in purulent infection? Arrest in the action of the morbid cause, and sufficient capacity of resistance on the part of the patient.

From this statement arise two leading indications of treatment: *first*, to diminish, and if possible, suppress the source of the infection; and, *second*, to supply the patients with necessaries for the struggle.

In discussing the theories of purulent infection, I have, as I proceeded, discussed all its conditions. There now, therefore, only remains for me to present a dogmatic summary of its etiology.

Gentlemen, you recollect that at the commencement of these lectures, I laid before you my views regarding the disposition to make pus which is observed in some patients, particularly in recently delivered women. There is often nothing to indicate the existence of this special predisposition; and women during the whole puerperal state, especially after delivery, have this purulent predisposition in a more marked degree than at any other time. In them, every inflammation is apt to pass into suppuration, as is seen by the course of their peritoneal, pleuritic, phlegmonous, and arthritic attacks. In such persons also, when inflammation attacks the venous system, it is not unusual to discover pus in the veins.

Before there can be purulent infection, there must be a wound, and it is also generally required, that veins of a certain size should be involved in the wound; but then the primary essential state is phlebitis, with the possibility of pus passing into the blood. Pus is seldom absorbed from the surface of wounds by large veins; but it may sometimes take place in suppuration of the placental wound. In that particular case, there is a special organic structure which may keep the uterine sinuses open. Still, that mode of purulent absorption is necessarily exceptional; for if the sinuses were always open, there would be uterine hæmorrhage. I am rather of opinion that in this class of cases, there is inflammation of the sinuses, and that this inflammation may give rise to infecting phlebitis. During the first days after delivery, the placental wound is rough with small projections constituted by the free extremities of the intra-venous clots, the length of which seldom exceeds one centimeter: these clots show a remarkable tendency to have their placental extremity purulent, and their intra-venous extremity cruoric: they are adherent to the internal surface of the sinuses, and are kept in place externally by their relation with the placental surface.

When the course of events is normal, the placental portion of the clots is eliminated, and the venous portion is absorbed: while at the same time, the vessels diminish in calibre, under the influence of the progressive and continuous contraction of the womb. But if suppurative inflammation affect the walls of some of the sinuses, there will be reason to fear infecting uterine phlebitis.

I have already said that what takes place within the uterine sinuses, in the situation of the placental wound, also occurs on the surface of wounds uniting by the first or second intention, though in a manner less evident, in consequence of the veins being there less numerous. If the wound heal by the first intention, there will be adhesive phlebitis, or merely an effusion of plastic lymph sufficient to establish adhesion of the walls of the veins. Should the wound suppurate, phlebitis is then necessary, and should the phlebitis be suppurative beyond the wound, there will be reason to dread purulent infection.

Ribes, Neucourt, and Velpeau regard erysipelas as an inflammation of the radical veins of the skin. As you know, erysipelas of new-born infants is prevalent during epidemics of puerperal fever. Again, both traumatic and spontaneous erysipelas is specially prevalent when surgical wounds complicate purulent infection. If then, we observe not only the simultaneous occurrence of erysipelas and purulent infection, but likewise their fellowship at a determinate period, are we not somewhat warranted to inquire, whether there does not exist in the atmosphere a morbid germ, which being deposited on the umbilical, or placental, wound, or on any surgical wound, will produce in one case phlebitis, and in another erysipelas, with or without purulent infection?

According to M. Charles Robin,[1] the pus-globule is simply a leucocyte possessing no particular property; and according to him, it is the purulent serosity which gives infectious properties to pus. If it be admitted, that the serosity of normal pus modified by a germ, a morbific spore, floating in the atmosphere, is of itself sufficient to give rise to purulent infection, that serosity will then be held to have the power similar to those serosities which are charged with a special virus, such as variola, vaccinia, or glanders, and the minutest wound will suffice for the absorption of this new poison.

Absorption of the thus modified serosity will explain the cases in which purulent infection is produced with its usual characteristics without any trace of suppurative phlebitis being found; just as the hypothesis of a morbific ferment floating in the atmosphere explains the epidemics which simultaneously strike down amputated patients and lying-in women. The works of Pasteur,[2] Reveil,[3] Chalvet,[4] and d'Eisell, show the very probable correctness of this hypothesis.

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[1] Robin (Charles):—In the *Journal de la Physiologie*, for 1859, p. 62.

[2] Pasteur:—*Annales de Chimie et de Physique*.

[3] Reveil:—Des Désinfectants, et de leurs Applications à la Thérapeutique; Paris, 1863.

[4] Chalvet:—Des Désinfectants, et de leurs Applications à la Thérapeutique et à la Hygiène. [*Mémoires de l'Académie Impériale de Médecine;* 1863: T. XXVI.]

M. Pasteur has shown, on the one hand, that the real ferments are organised bodies, the aliment of which is albuminoid matter. He has shown, on the other hand, that the dust floating in the atmosphere contains starch, and vegetable and animal spores, capable of attaining life. The same philosopher has made it appear, that special spores are necessary for the production of special fermentations; and that these different ferments are recognisable by their form. The spore is an organised, living cellule vegetating upon the elements which surround it: but to enable this vegetation to take place, it is necessary that certain determinate conditions exist in the medium in which the germs are placed. When you have once determined these conditions, you may, by employing the spores of the alcoholic, acetous, and lacteous ferments, obtain respectively the alcoholic, acetous, or lacteous fermentation.

Here, then, is the great theory of ferments, by which fermentation is ascribed to an organic function. Every ferment is regarded as a germ, the life of which is manifested by a special secretion.

Perhaps, this is so likewise in respect of morbid viruses. Possibly, they are ferments deposited in the organism, which at a given moment, and under certain determinate circumstances, make themselves known by the multiplicity of their products. Thus, the variolous ferment will give rise to the variolous fermentation, in the midst of which a profusion of pustules will appear: so is it with the viruses of glanders and tag-sore.

Other viruses seem to have a local action; but in course of time, they do not the less modify the entire organism: as examples, I will mention hospital gangrene, malignant pustule, and contagious erysipelas. Under these circumstances, may we not admit, that the ferment, or organised matter of these viruses is transported to one place by the lancet, and to another by the air or the dressings?

These statements are not mere hypotheses: chemical analysis and the microscope prove the existence of morbific dust in hospital wards. Thus, M. Chalvet, in his interesting researches into the causes of the insalubrity of hospitals, found that the air of the Hôpital Saint-Louis contained a large quantity of starch-corpuscles: besides, on the walls, window-frames, curtains, and beds, there was found, by the aid of the microscope, a great quantity of putrescible organic matter. It has been stated by the same observer—and I have lately had the opportunity of confirming his remark—that dressings returned from the lavatories have been found still soiled with organic detritus, with linseed, and with spots, recalling the uses to which they had been formerly applied. Might not these linens, stained with blood and altered pus, be vehicles of contagion? Was it not an old custom to preserve vaccine matter on linen and cotton threads? Imagine the consequence of using the imperfectly washed linen of small-pox patients!

M. Chalvet has also stated, "that the vapor of water condensed near a source of suppuration, is, prior to the dissemination of miasmata, greatly charged with irregular corpuscles, in all respects resembling dried pus. It is not unusual to find there some fragments of the colouring matter of the blood."[1]

Eiselt (of Prague) states that he saw small pus-cells in the air of a ward occupied by patients with epidemic purulent ophthalmia.

On this subject, M. Chalvet, in reporting to me the result of his experiments, thus expressed himself:—

"The *nosocomial atmosphere* has now ceased to be a term without meaning: the air of an hospital differs so essentially from pure air, that attention to the fact can no longer be neglected. Since 1860, I have had an opportunity of witnessing the experiments of M. Reveil, (quoted by M. Devergie); and have satisfied myself, in the most positive manner, that organic corpuscles were present on the platinum plates of the apparatus constructed by that chemical *savant*. I saw on them principally cells, débris of epithelial cells, corpuscles of different forms (which assumed a yellow colour under the action of nitric acid), and shreds of charpie charged with the same organic corpuscles.

"On one particular occasion, I saw, along with M. Kallmann, in M. Reveil's laboratory, incrusted organic débris, and a granular substance which produced a reaction with copper. The dust examined had been collected in an ophthalmic ward where the sulphate of copper was extensively used as a caustic.

"The dust dusted off the walls [*par l'époussetage sur les murs*] of Saint-Augustin's ward, in M. Richet's service at Saint-Louis, yielded thirty-six per cent. of organic matter in my first analysis. Dust taken from the same walls upon another occasion, and analysed in M. Reveil's laboratory, yielded forty-six per cent. of organic matter, consisting chiefly of epithelial cells exhaling the odour of burnt horn. On moistening the dust so collected, a strong putrefactive odour was exhaled. No doubt, the vast coat of mixed dust which clothes the rarely cleaned walls of the wards in the old hospitals, has the power of generating gases calculated to favor the aerial transport of bodies which perhaps play an important part in the constitution of the nosocomial atmosphere."

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[1] CHALVET:—Op. cit.

These facts are, in my opinion, very significant. There has always existed, from reasoning, a strong disposition to attribute much in the spread of endemics and epidemics, to the transport of morbific molecules; and now, the same conclusion has been arrived at from direct examination of the atmosphere. With some justice, M. Pasteur remarks:—"It would be very interesting to compare the organised bodies disseminated in the atmosphere in the same place at different seasons, and in different places at the same period. It appears to me, that we would gain an increased knowledge of the phenomena of morbid contagion, particularly during the progress of epidemics, by researches pursued in that direction."

The germs which may exist in the atmosphere at a given time, under circumstances to be determined by future inquiry, will not become developed with equal facility in all patients, because persons differ exceedingly in respect of the condition of receptivity: certain organisms, like certain soils, will not accept certain germs. Throughout a country, the winds have diffused the same seeds; and yet the same seeds do not spring up everywhere. In one situation, the ground is too wet, and in another it is too dry: in some places other seeds having already grown up, the new seed is choked: or perhaps, the seed springs up wherever it falls: in one place there is too much light and too much heat: in another place the temperature is too high, while in another it is too low, so that here the plants are puny, and there they are strong.

Before inquiring, whether purulent infection can be foreseen or arrested, let me speak to you of the state of the blood.

The blood, in purulent infection often presents a brown chocolate color. In different parts of the venous system, especially in the right side of the heart, there are cruoric clots of the consistence of pitch, in the midst of which are seen small whitish masses, which might be mistaken for coagulated fibrin. Many observers have been nearly certain that these *post mortem* modifications in the consistence and color of the blood were attributable to the presence of pus in the blood. Recently, M. Donné, and afterwards, M. Bouchut published cases in which they made out, that in purulent infection, we can recognise the presence of pus in the sanguineous fluid: in fact, they described bodies identical in dimensions and in microscopic appearances with pus-globules properly so called. It appears then, that the presence of pus in the blood may sometimes be detected, but that the search for it is often fruitless.

Some micrographers, however, hold, that the supposed pus-globules are only the white globules of the blood, which, under certain conditions, unite and become deposited after death, in small masses, within the sanguineous clots.

In cases of purulent infection following delivery, I have observed —as had already been noticed in cases of suppurative phlebitis— that the blood of the inflamed veins had a chocolate color, like the color of the blood of patients affected with leucocythæmia: this coloration of the blood was less and less marked, the greater its distance was from the seat of suppuration in its onward passage to the heart.

Several times, I have detected pus mixed with blood in the hypogastric and common iliac veins, up even to the vena cava inferior. Note well, that in these cases there was no obliterating clot.

At other times, this purulent mixture, verified by microscopic examination, was in the ovarian veins, and I have demonstrated it in the left ovarian vein, up to its junction with the emulgent vein. Thus, one could follow step by step, so to speak, the gradual decrease of this special coloration of the blood, up from the uterine veins filled with pus to the vena cava inferior or emulgent vein. Then, if we at the same time examine with the microscope the blood supposed to contain pus, and the purulent fluid of suppurative phlebitis observed in the same subject, we find in both absolutely identical pus-globules: these globules, however, are fewer in number, the more remote from the seat of suppuration is the place from which the blood is taken. The fact was observed by Hunter.[1]

The examination, then, of the blood after death leaves no doubt in my mind. I am convinced that it can be shown, after death, that the blood of patients who have died with purulent infection, is contaminated by pus in varying proportions.

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[1] Hunter:—Œuvres Complètes; traduites par G. Richelot. Paris, 1843.

But M. Charles Robin has come to the conclusion, that the white globules may be formed in every part of the organism, and that there is no difference between the leucocyte and the pus-globule. M. Robin thinks that leucocytes give pus its color, but not its nature, the latter being due to the fluid of which it is principally composed. He holds that pus derives its essential characters from the serum, and not from the leucocytes.

This assertion involves two allegations; viz. that the pus-globule and the leucocyte are identical, and that the special nature of pus is derived from its serosity.

In respect of the identity of the pus-globule and the leucocyte, I must say, that I have several times examined the blood of patients affected with diseases in which M. Robin has pointed out the frequency and comparative identity of leucocytes, and that I have sometimes met with very decided differences, a circumstance which I cannot omit mentioning—for example, in the puerperal state, in confluent and non-confluent small-pox, in paludal cachexia, and in purulent infection.

In all the patients embraced in this comparative study, the blood was taken from a prick at the end of one of the fingers, and was received on glass plates. On every occasion, the microscopic examination of the blood was made quite close to the patient's bed. Here are the results of my observations:—

In a little child, who presented clinically all the symptoms of hæmorrhagic leucocythæmia, I found a very great number of large white globules, from twenty-seven to thirty white globules to each preparation. Each of these leucocytes, being fixed on the field of the microscope, was found to measure ten, twelve, or thirteen thousandths of a millimeter by Nachet's micrometer, using a magnifying power of five hundred and eighty diameters. The leucocytes were formed of a cell containing numerous nuclei. The red globules were of natural form, and of the normal dimensions, varying from the six to the seven thousandth part of a millimeter.

The blood of several small-pox patients was examined at the time the eruption was coming out, and also during the period of suppuration. The pus-globules were identical in dimensions and appearance with pus-globules taken from the pustules: these globules did not measure more than the six, seven, or eight thousandth part of a millimeter: there were from six to twelve of them in the field of the microscope, and it was with great difficulty that I detected on some of the preparations one or two white globules of from ten to twelve thousandths of a millimeter.

In a woman, who died after presenting the symptoms of purulent infection, examination of the blood before and after death, showed a great number of pus-globules, and very few large white globules, in the blood. It does not seem necessary to give you an abstract of my other similar, and similarly conducted, observations. I only wish to remark, that I consider that these inquiries seem to have established the existence, in certain cases, of pus-globules properly so called, and that the large white globules were few in number. I must add, however, that in other researches, I observed a greater number of white globules, both in small-pox patients and in lying-in-women suffering from purulent infection.

Great consideration is due to the importance accorded by M. Robin to the serum of the pus. That importance has already been manifested in the case of the virulent pus of variola and glanders, the respective specific differences of which have been demonstrated by inoculation. These differences also certainly exist in respect of benignant and malignant pus; that is to say, between pus the serosity of which when absorbed, does or does not produce general infection. The considerations into which I entered, when speaking of wounds modified by the germs or specific spores, seem to prove that these differences exist.

In purulent infection, has the serosity of the pus the power of transforming blood into pus? I need not repeat the assertion of Hippocrates, Galen, Van Swieten, and De Haen, whose idea was that the pus, diffused throughout the organism, engenders pus at the expense of the humors. Looking to the great rapidity with which pus is diffused throughout the entire organism in cases in which there is no suppurative phlebitis, one is constrained, I think, to attribute a very large share to the absorption of the serosity when numerous metastatic abscesses are formed.

To sum up:—I consider purulent infection to be the result of poisoning of the blood with pus.

Suppurative phlebitis generally furnishes the pus, which sometimes passes directly into the venous sinuses and plexuses, although they have not been the seat of any suppurative inflammation. Ulceration of the arterial walls, and particularly of the aorta or sigmoid valves, may be the source of purulent infection, as has been shown by Dr. Leudet of Rouen, in a memoir published in the *Archives Générales de Médecine.* But although purulent infection is most frequently the consequence of suppurative phlebitis, there are some epidemics in which we must, perhaps, seek for the cause, not in crowding, but in a special state of the atmosphere, in its containing, at a given time, altered pus-globules, which, being deposited on the wound, act in such a manner on the serosity of the pus of the wound as to modify it specifically, and give it the power to engender purulent infection of the system.

The serosity of the wound thus modified, may, in respect of its immediate and remote action, be compared to a virus.

My view is, that pus makes pus, and that putridity makes putridity, just as variola, syphilis, and glanders make variola, syphilis and glanders.

But that pus should make pus, it is necessary that the pus be of a certain nature; and probably, it is in the serosity of the pus that we must search for the material specific differences; for according to M. Robin, it is in the serosity, and not in the globule of virulent pus, that the specific virus resides. From this point of view, all pus is looked upon as having a common element—the white globule; and also, a special element—the serosity. We are ignorant of the special principle of the serosity: what we fear is its absorption. Can we oppose a barrier, and dry up the source of infection?

I am now naturally led to speak of the *treatment* of purulent infection. But here, let me say, that my object in discussing so fully the doctrinal question of purulent infection in general has been for the purpose of throwing light upon *puerperal* purulent infection in particular. I leave you, therefore, to learn from systematic authors on external pathology the means to be used for the prevention of phlebitis, that too fertile source of purulent infection.

I believe that hospital hygienics embrace means by which the germs of infection may be neutralised, and its progress modified, should the germs become developed.

There is no specific cure for purulent infection. One ought not, however, to neglect the use of means, which, by being directed to the entire system, may determine salutary crises, and sustain the strength of the patient. Thus, sulphate of quinine may be successfully employed to check the periodic recurrence of the paroxysms of fever; but this medicine will not be any more useful than tincture of aconite in staying the progress of the infection, once pus has become mingled with the blood. The efforts of the physician ought to have another aim: his aim ought to be, to study the curative proceedings of nature in cases of spontaneous cure, endeavouring to imitate, or better still, to second her efforts to eliminate the morbific principle. In sketching the progress of purulent infection, I mentioned, that many patients had profuse viscid sweating and severe diarrhœa: I mentioned that the sweat and the diarrhœa present characters more easy to verify than to describe, but that their dominant characteristic is a special odour. These cutaneous and intestinal evacuations may be considered critical; and they ought to be promoted by sudorifics and purgatives, in the hope of obtaining results equally favorable with those obtained by Sanson and Vidal. It is useful to employ, at the same time, slightly stimulating stomachic beverages, and to place the patients under the best hygyenical conditions.

To bring these remarks to a close, I shall now state my conclusions.

Purulent infection never occurs unless there be a wound. A wound is the essential obligatory condition. Every wound may have suppurative phlebitis as a sequel. Suppurative phlebitis pours pus *en nature* into the circulation. Perhaps this takes place continuously, although the intermissions in the rigors seem to indicate that the poisoning only takes place in an intermittent fashion.

Purulent infection may also originate in abscesses of the heart and aorta. This form of infection is rare.

Capillary phlebitis may induce infection by leading to the formation of pus; but in epidemics of purulent infection, the serosity of wounds, specially modified by atmospheric conditions, may be absorbed by the capillaries, without there being any ossicula or erosions of the vessels; and infection is the consequence of this absorption. *The serum of the pus acts in a manner similar to the virulent inoculable serosities.*

Two principal indications ought to be fulfilled with a view to prevent or arrest infection. The first consists in so acting on the wounds as to prevent the occurrence of suppurative phlebitis, or interpose an obstacle to the absorption of infectant serosity. The second indication, based on the progress of the infection and the study of the crises, consists in the use of such means as promote and maintain the curative crises.

In conclusion, it is the duty of the physician, to place his patients under the most favourable hygienical conditions, which can be obtained, and thus enable them to struggle sufficiently long to finally triumph over the infection.

LECTURE XCV.

PHLEGMASIA ALBA DOLENS.

Phlegmasia in Recently Delivered Women.—Phlegmasia in Cachectic, Tuberculous, and Cancerous Subjects.—Semiotic Value of Phlegmasia in Cachectic Diseases.—Phlegmasia in Chlorosis.—Phlegmasia in Recently Delivered Women: 1st by Spontaneous Coagulation: 2nd Consecutive upon Uterine Phlebitis.—Symptoms of Phlegmasia: pain, œdema.—Venous Cords.—Collateral Circulation.—Temperature of the Affected Limbs.—Absence of Lymphangitis and Adenitis.

Gentlemen:—Those of you who attend my clinical service must have remarked the frequency of the occurrence of phlegmasia alba dolens, an affection quite special, and well deserving attention from the numerous circumstances under which it is observed. You recollect that we have studied painful white œdema, not only in recently delivered women, but also, and more frequently, in persons of both sexes affected with pulmonary phthisis or internal cancer. To-day, I propose to speak to you of this affection which always has, as its primary cause, a special alteration of the blood, an alteration which exists in the puerperal state, and in many cachexiæ. I shall not attempt to establish on a statistical basis, the relative frequency of painful œdema in the cachexial and puerperal state: I merely wish to remark that this affection is frequently observed, and that, independent of the puerperal state, it may become a valuable element of diagnosis.

It is one of those diseases which demand minute study, because doubts still exist as to the nature of the affection, notwithstanding the frequent opportunities which occur of observing it. Painful œdema is a disease the nature and etiology of which have been differently explained. I must, consequently, state, in detail, the cases which will serve as the basis of the general descriptive sketch, which I am about to place before you. I must also carefully recall to your recollection the anatomical details, because these details will have great value when we proceed to fix the precise seat of the affection. When the seat of the disease is well understood, you will be the better able to understand, with the aid of anatomy, the symptomatology of the disease, and the complications, sometimes very serious, to which it gives rise.

A woman, 33 years of age, was admitted to Saint-Bernard's ward, presenting all the signs and symptoms of the third stage of pulmonary phthisis—amphoric blowing, gurgling, pectoriloquy, purulent expectoration, great emaciation, profuse sweating, hippocratic fingers, hectic fever, functional derangement of the stomach and bowels, ulcerative laryngitis, and dysphagia. The patient had been six weeks in my wards when I ascertained she had white œdema of both superior extremities, involving the whole of the left arm, but limited in the right arm to the region of the elbow. On the first day, I searched, but in vain, for an obliteration of the superficial veins of the arm: I then thought that the deep veins were the seat of a spontaneously formed coagulum. The patient had not experienced any pain in the regions affected with œdema; and to cause suffering, it was necessary to compress the limb, either in its entire circumference, or over the course of the deep venous branches. Next day, the superficial veins were seen, blue and turgid, through the skin: they then seemed to be very probably the seat of a collateral circulation, rendering it still more probable that there was obliteration of the deep veins. During the first few days, I did not observe a fact which I afterwards noticed, and which must be added to the above description, viz. that on the inner surface of the arm, along each side of the humeral artery, a hard cord could be felt.

Soon afterwards, on each fore-arm, the superficial radial veins were felt to be obliterated, the skin was observed to be red in the course of the veins, and there was slight pain on pressure in that situation. The œdema of the fore-arms slowly decreased, till it quite disappeared, while, at the same time, the bulk of the superficial veins diminished, they recovered their elasticity, and again became permeable, as was clearly proved by the effects of compression made above and below the affected parts: when pressure was made near the elbow, the vein became dilated, and when made in the inferior third of the fore-arm, the vessel became quite empty. From the

31st January to the 14th February, 1862, I had the advantage of being a clinical witness of the formation and disappearance of the clots by which the superficial veins of the fore-arm were obliterated. Probably, a similar process took place in the deep veins of the same regions, in proportion to the progressive diminution of the œdema and pain.

From the 12th February, both legs became the seat of œdema, extending upwards from the feet, and soon invading the thighs. On the first day, this œdema was especially marked in the left limb, where pressure with the finger left its print and caused pain. In the upper part of the leg, one could feel a hard superficial vein, which became lost in the popliteal cavity. The internal saphena vein was distended with fluid blood; and on exploring the femoral vein in its superior third, that vessel was found to be knotty, hard, and painful.

The right leg and thigh were œdematous, but in a less degree: the superficial circulation was manifested by the blue color of the veins, which seemed more than normally numerous: a hard painful cord, the obliterated femoral vein, was felt in the triangle of Scarpa.

On the following days, the œdema of both legs increased, but always continued greatest in the left. In that limb, where the œdema was greatest, there was soon perceived complete obliteration of the internal saphena vein up to its junction with the femoral vein, while only the lower third of the right saphena was hard and knotty. By compression at the lower third of the thigh, it could be positively ascertained, that the circulation was still possible in that situation. There likewise appeared some days later, in the legs, thighs, and round the knees, numerous venous capillaries, forming isolated groups, and apparently developed on the surface of the dermis. Some of these groups were red, others were white, and others again, were the seat of obliterated capillaries, quite appreciable by the finger: and when these vascular groups were subjected to pretty strong pressure, pain was occasioned.

Do you not observe in this last statement, distinct evidence of a well-marked tendency to the re-establishment of the circulation. The deep veins were seized in the first instance, the saphena veins supplied their place till they themselves become involved in the obliterative process: the capillary vessels then became very visible from congestion, and were invaded by obliterative clots, while the œdema went on constantly augmenting. The pain, limited in the first instance to the course of the principal vessels, soon became general, both on the surface and in the deeper parts: the skin in the situation of the capillary groups became very painful. In some places, where no trace of vessels could be discovered, light quick rubbing with the pulp of the finger occasioned pain.

Eleven days after the commencement of these symptoms, the œdema of the lower extremities had greatly increased: this was particularly the case with the left limb, where the saphena vein, and the posterior superficial vein of the leg, were decidedly hard: there was a little redness in the calf, where the acute character of the pain led one to suppose that there was inflammation of the vein in that situation. All the superficial veins of the right leg were greatly distended: on palpation, no clots could be recognised: for several days the right limb remained in the same state, while in the left leg the œdema increased, and the superficial veins became more and more painful in the popliteal space and in the whole course of the internal saphena vein: the skin in these situations presented an erysipelatous redness.

On the eighteenth day, the left leg, and particularly the foot, had a notable lividity, where the slightest rubbing was exceedingly painful. The superficial abdominal veins were very much injected. There was no pain in the groin, nor in the left iliac fossa. On several places of the left leg and thigh, there was slight marbling of the skin and true ecchymosis; and in these situations, pressure caused pain. Similar phenomena were soon observed in the right limb; and the skin was figured with numerous capillary veins.

The œdema continued in the same degree. There was acute pain in the region of the liver. On the twenty-second day, the patient was seized with diarrhœa; and died without presenting any notable disturbance of the brain, lungs, or heart.

The autopsy was very interesting. It afforded opportunity of studying the obliterative clots in the situations where they still existed, although the œdema had continued till death; it likewise afforded an opportunity of studying the successive modifications which some of these clots had undergone; and also, the state of the parietes of the vessel where the circulation was re-established.

In the left limb, the veins of the calf, the femoral, the internal saphena, and the external iliac, were obliterated by fibrinous clots the highest of which was situated at the opening of the hypogastric into the common iliac vein. There was found at that point a clot, rounded in form at its free surface, and bestriding, so to speak, a spur formed by the hypogastric and external iliac veins, with which its lower portions were continuous. The terminal clot was not adherent to the walls of the vessel: its form was cylindrical: its upper portion was rounded and devoid of any rent. It was composed of concentric layers, softened in the centre, so that portions were liable to give rise to pulmonary embolism by being drawn into the torrent of the circulation flowing from the hypogastric vein.

In the right limb, the clot, the ramifications of which occupied the deep femoral veins, the popliteal and tibial veins, stopped short where the internal saphena opens into the femoral: at that point, the clot was fibrous, similar in appearance, form, and structure, to that of the common iliac vein of the left limb. The clot contained in the saphena vein was cruoric, of recent origin, and non-adherent to the parietes of the lower veins.

This autopsy shows clearly that the continuance and extent of the œdema of the lower extremities was explained by the continuance, extent, and structure of the clots.

This is not all:—The examination of the veins of the superior extremities, ought, still farther, to demonstrate the correspondence of the anatomical appearances after death, with the symptoms during life. During life, there was a partial phlegmasia of the superior extremities, the commencement, progress, and termination of which coincided with the appearance, duration, and disappearance of the coagula in the superficial veins. At the time of the appearance of the œdema, there was induration of the brachial vein in both arms, and of the superficial radial vein in both fore-arms: in the course of these vessels, a red line, pain, and swelling were observed; and under the finger, there rolled a cord, the limits of which could be easily determined: at the same time, there was partial œdema:—little by little, the œdema disappeared, and proportionately to its disappearance the pain, swelling, and hardness of the vein went away. It was ascertained, by anatomical examination, that there was no trace of inflammation remaining in, or around, the veins: the veins contained no obliterative clots, and were free: their walls had regained their normal elasticity. It was only in the left brachial vein that there was a fusiform cruoric clot, fringed at both extremities, adherent only at some points of its surface, and sufficiently small to allow the circulation to go on round it. It is probable, then, that the clots by which the veins had been formerly obliterated, had been absorbed *in situ*.

The case is a rare example of general intra-venous coagulation in the four extremities. Under what conditions does the blood present this tendency to spontaneous coagulation? Gentlemen, you are aware that in the cachexiæ generally, and in the tuberculous and cancerous cachexiæ particularly, the blood has undergone important changes. The beautiful works on hæmatology of MM. Andral and Gavarret[1] and MM. Becquerel and Rodier leave no room for doubt on that point: the modifications consist chiefly in a change of the proportions of the constituents of the blood: thus, in every cachectic affection, there is a diminution of the red globules, and an augmentation of the fibrin and serum. Now, the blood having a great tendency to spontaneous coagulation, we are entitled to ask, whether this coagulation be not due to an excess of fibrin, or to the fibrinogenous element?

One word, Gentlemen, on this element:—You know that in serous effusions into the pleural cavities, there exist two isomeric substances, fibrin and albumen. When serous fluid which has been effused into the pleuræ is exposed to the air, coagulation of the albumen does not take place at a temperature under 70° or 75° centigrade, while there is spontaneous coagulation of the fibrin some degrees below zero. Moreover, when we extract the spontaneously coagulable fibrin, either by heating or straining through a linen cloth, and leave the remaining serosity exposed to the air, we are surprised to see, that, some hours after the first straining, a new fibrinous coagulation has formed in the fluid. It is evident, therefore, either that all the fibrin has not been removed in the first experiment, or that the fluid contains a special substance which may at a given time present all the characters of fibrin. To a substance of that character, Virchow has given the name of "fibrinogenous substance," which might be more properly designated *inogène*, that is to say a substance capable of giving birth to a new quantity of fibrin. Perhaps this substance exists in serum of the blood of cachectic persons; and if so, its coagulating properties explain the tendency to spontaneous coagulation in the blood of cachectic subjects.

The fact, long well known, that the blood of cachectic persons has a strong tendency to coagulate, explains the frequency of coagula in the vessels of tuberculous patients who have reached the cachectic stage of their disease. I wish also to recall to your recollection some of the facts which prove that the same spontaneous coagulation is common in cancerous patients, and gives rise to *phlegmasia alba dolens*. In Saint-Bernard's ward, you have had frequent opportunities of observing this phenomena in women affected with cancer of the uterus. Patients of this description, in the last stage of their disease, present the symptoms of cachexia, and then, all at once, the inferior extremities become swollen, soon after which the saphena and crural veins can be felt to be hardened: in such cases, it is found, on examination after death, that their hard condition is owing to cruoric or intra-vascular fibrinous clots.

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[1] ANDRAL AND GAVARRET:—Recherches sur les Modifications de Proportion de quelques principes du Sang dans les Maladies, Paris: 1842.

I have long been struck with the frequency with which cancerous patients are affected with painful œdema in the superior or inferior extremities, *whether one or other was the seat of cancer*. This frequent concurrence of phlegmasia alba dolens with an appreciable cancerous tumor, led me to the inquiry whether a relationship of cause and effect did not exist between the two, and whether the phlegmasia was not the consequence of the cancerous cachexia. I have since that period had an opportunity of observing other cases of painful œdema, in which, at the autopsy, I found visceral cancer, but in which during life, there was no appreciable cancerous tumor; and in which there existed a cachexia referable neither to the tubercular diathesis, the puerperal state, nor chlorosis. I have thus been led to the conclusion, that when there is a cachectic state not attributable to the tuberculous diathesis nor to the puerperal state, there is most probably a cancerous tumor in some organ.

Many of you remember the case of a patient in the wards of Legroux, my late lamented colleague. The man to whom I refer was fifty-nine years of age. Without any known cause, he was seized with phlegmasia alba dolens of the left leg. The case presented all the characters of phlebitis: there were acute deep-seated pains in the calf, beginning about the lower third of the leg and extending up to the hamstring. The patient was extremely pale, and had a general cachectic hue. Legroux was inclined to think that the patient had leucocythæmia. When consulted, I said:—"Perhaps, this patient has leucocythæmia; but he has phlegmasia alba dolens, and consequently deep seated, concealed cancer."

I sought for this cancer with the greatest care: and during the six weeks which the patient remained in the wards of Legroux, that physician searched for it with his habitual scrupulous attention, and yet was unable to detect the signs of a cancerous affection. At the autopsy, there was found an anular cancer of the pylorus, which, as it allowed the passage of the food into the duodenum, had not given rise to vomiting of any special character.

In other cases, in which the absence of any appreciable tumor made me hesitate as to the nature of a disease of the stomach, my doubts were removed, and I knew the disease to be cancerous, when phlegmasia alba dolens appeared in one of the limbs.

Some years ago, one of the professors of the Faculty of Medicine had symptoms of simple ulcer of the stomach. Several physicians had been consulted; and as they found no tumor in the region of the stomach, they were disposed to regard the vomiting as symptomatic of simple ulcer. Soon after this, I learned that the professor had phlegmasia, whereupon I unhesitatingly declared that he would sink under advancing cancerous disease: the rapid progress and fatal issue of the case proved my diagnosis to be correct.

In 1860, a man, about forty years of age, consulted me, as a private patient, regarding pain and a feeling of weight in his left leg. On questioning him, I learned, that at a previous period, he had experienced similar pain in the right leg; and that he had, subsequently, been operated on by M. Maisonneuve for a tumor of the testicle. To me, these facts were of the greatest importance: from them, I concluded, that the patient had had previously phlegmasia, symptomatic of a cancerous tumor of the testicle, and that at the time when I was consulted, the œdema of the left leg was caused by a deep-seated cancerous affection: in point of fact, by palpation, I detected within the abdomen, tumors, of the cancerous nature of which I had not the least doubt.

We must not suppose that painful œdema of the inferior extremities in cases of cancer of the testicle, uterus, or rectum, results from the inflammation of the veins of the primarily diseased parts being propagated to the deep-seated veins; nor are we to believe that the œdema is the mechanical consequence of pressure exerted on the abdominal veins by tumors or diseased glands. Such opinions are untenable, for whoever carefully analyses cases will find, that cancerous tumors of the stomach or breast give rise to this kind of phlegmasia. I might cite many cases in confirmation of this statement; but will be satisfied by referring to the case of a man aged forty-six, mentioned by Virchow, who had carcinoma of the stomach coexisting with double phlegmasia of the lower extremities and painful œdema of the left arm.

So great, in my opinion, is the semiotic value of phlegmasia in the cancerous cachexia, that I regard this phlegmasia as a sign of the cancerous diathesis as certain as sanguinolent effusion into the serous cavities.[1]

In the cachexiæ, as I have told you, there exists a special crasis of the blood, which, irrespective of inflammation, favors intra-venous coagulation. This state of the blood is likewise met with in chlorosis properly so called, and in the puerperal state.

Painful œdema, as an epiphenomenon of chlorosis, is, however, a rare affection. I must describe a case which occurred in my wards, and was reported in 1860 by Dr. J. Werner in his inaugural thesis. That young physician particularly calls attention to phlegmasia as an epiphenomenon of cancerous cachexia.

A young woman, aged twenty-five, was admitted to my wards with all the signs of chlorosis—extreme pallor, blowing sound in the vessels of the neck, palpitation of the heart, intercostal neuralgia, dyspepsia, and amenorrhœa. She was forthwith treated, a little actively, when, all at once, she experienced pain in the left inguinal region; and on the same day, there was detected phlegmasia of the right inferior extremity, characterised by œdema of the limb and intra-venous coagulation. After continuing for three weeks, the œdema disappeared.

Painful œdema was formerly looked upon as an affection peculiar to lying-in women: hence, it received many names referring to the state of the recently delivered woman. Mauriceau, Puzos, Callisen, and White have devoted special chapters to the swelled leg of lying-in women, to lacteal engorgement of the inferior extremities, and to the *phlegmasia alba dolens puerperarum*; but Robert Lee and White were the first to describe the lesions of the veins which accompany painful œdema. Subsequently, MM. Bouillaud and Velpeau, in works published in 1823 and 1824, had the merit of showing the part which obliteration of the veins has in producing partial dropsies. Later researches confirmatory of the statements of these savants, have demonstrated that obliteration of veins may occur spontaneously, or, in other words, may depend upon a crasis of the blood, and not upon inflammation of a vein.

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[1] See Lecture XXXII: on Paracentesis of the Chest: Volume Third, p. 259.

The remarks which I have already made upon the frequent occurrence of phlegmasia in phthisical and cancerous patients, render it unnecessary for me now to prove to you that this kind of œdema is not peculiar to lying-in women; and that there exists in the cachexiæ, as well as in the puerperal state, a particular condition of the blood which predisposes it to spontaneous coagulation, that is to say, a condition in which there is an excess of fibrin, a diminution of blood globules, an increase of water, and an increase of white globules.

I have told you, that the chlorosis of pregnant women is very seldom complicated with phlegmasia; and that the œdema of pregnant women is not due to the coagulation of the blood, but results from a double condition, the aqueous state of the blood, and the embarrassment of the circulation in the inferior extremities, mechanically produced by the augmented volume of the uterus. When anasarca occurs in a pregnant woman, we have to fear another complication—albuminuria.

Though painful œdema is never observed in the pregnant, it is often seen in the recently delivered woman; but its determining cause cannot always be discovered. We may meet with this œdema in cases in which neither the duration of labour, nor the presentation have been abnormal; and in which there has been no need of any important obstetrical manipulations. On the other hand, in cases in which partial œdema results from phlebitis, we can often trace back the affection to the local cause in which it had its origin. Uterine phlebitis may be propagated from the uterine veins to one of the hypogastric veins, and from the latter to the common or the external iliac vein. At autopsies, I have frequently recognised this causation, have seen obliterative phlebitis, and have traced the obliterative clot into the iliac veins, and sometimes even into the inferior vena cava. So long ago as 1826, Velpeau published cases demonstrating the propagation of the inflammatory process from the uterine to the iliac veins.

In these cases, there is sometimes real inflammation, which becomes the cause of the œdema. We then discover that the uterine sinuses and veins contain pus, and have thickened walls: but besides the pus, we find that these vessels contain clots which present an obstacle to the production of purulent poisoning. These fibrinous clots become covered with new layers of fibrin, which, like ceaselessly formed strata furnished by the fibrin of the circulation, become so added to, and superimposed upon one another, that the clots are lengthened out to the junction of the hypogastric with the iliac vein. At the junction, the clot projects, and is, at that point, covered with a new stratum of fibrin, which augments little by little, till the iliac vein itself is soon partially or wholly obliterated. Adhesions form between the walls of the vessel and the newly formed clot: this is the time at which œdema of the entire right or left inferior extremity shows itself. It almost never happens that both limbs are simultaneously affected, that is to say, they do not both become œdematous on the same day, but first the one and then the other. This sequence of phenomena is fully explained by clinical study and necroscopic examination: either inflammation of the uterine sinuses is propagated to both iliac veins at an interval of some days occurring between the date at which each limb is attacked; or, the clot formed in the common iliac vein of one side is prolonged into the vena cava inferior, and from it, into the common iliac vein of the opposite side. The greater frequency of simple phlegmasia on the left side, and of double phlegmasia beginning on the left side has been attributed by anatomists to the mutual relations of the arteries and veins at the sacro-vertebral angle. You are aware that in that region, the arterial system is situated on a plane anterior to the venous system, so that both common iliac arteries pass in front of the veins of similar name, dividing them at right angles; again, before reaching the inferior vena cava, the left common iliac vein is divided, almost transversly, by the right iliac artery: from these relations, it results, that, on the dead body, the arteries leave their print on the subjacent veins; and it is not unusual, when these veins are filled with clots, to find these clots strongly depressed at the spot at which the veins are crossed by the arteries. This compression is specially manifest in the left iliac vein, and anatomists ascribe to the more frequent compression of the left iliac vein, the more frequent occurrence of phlegmasia on the left side. On the other hand, accoucheurs have supposed that, as left anterior occipito-iliac presentation is the most common, the greater frequency of phlegmasia in the left limb is perhaps owing to the pressure of the head during labor on the left iliac vessels. I do not reject this reasoning; but I only accept the anatomical conditions as a determining cause. The same conditions exist in nearly every recently delivered woman, or in all cachectic persons; and yet phlegmasia is not a necessary complication in either class of patients. Consequently, there exists a special cause, which is unknown.

Painful œdema generally shows itself in a sudden manner: without appreciable cause, the patients complain of pain in a limb, which is at the same time observed to be œdematous. The pain is not always of the same kind: it is sometimes a feeling of weight and distressing numbness of the entire affected limb: at other times, it is a constant pain, intensified at a particular place, which, in the inferior extremity, is generally the calf, the groin, the lower part of the thigh, or the popliteal space: in the superior extremity, it is the axilla. If pressure with the finger be made over the seat of pain, or if the muscular masses in which pain is felt be grasped with the hand, an increase of pain is produced, which is often so great as to cause the patient to cry out. Sometimes, the sensibility of the skin of the whole limb is obtunded: while, at other times, touching or rubbing it lightly will occasion great suffering. I have often observed this cutaneous hyperæsthesia, which, strange to say, is less under strong than under slight pressure. Sometimes, the pain and numbness are accompanied by inability to perform the least voluntary movement: for example, the patients can neither extend nor flex the toes, nor move the leg or thigh. Though in some cases there are articular pains to account for this immobility of the limbs, in others, in which pressure causes no articular pains, all movement is as impossible as if muscular paralysis existed.

An attempt has been made to give a special description of the œdema: it has been alleged that it shows itself first at the upper part of the limb, and then appears secondarily in the remoter parts. Gentlemen, I confess that I have never observed the affection to have that beginning or course: on the contrary, I have always seen partial œdema begin in the most remote and depending parts, subsequently gaining the upper parts of the limb. To be convinced of the truth of this remark, it is only necessary to observe with care the progress of the œdema in cases of double phlegmesia. Directing our attention to the possibility of the occurrence of double œdema, and continuing to observe daily the left limb still healthy, we will soon observe the œdema begin at the malleoli and instep: the skin will be seen to have acquired a dull color, and the subcutaneous cellular tissue will be seen to retain the print of the finger: then by slow degrees, the œdema will ascend, and soon the intracellular effusion will give a rounded form to the whole limb. The limb generally acquires the form of an elongated cone, the base of which is the root of the limb. This description applies to œdema consequent upon obliteration of the principal vein of the affected limb.

I have also seen cases of partial œdema resulting from the obliteration of secondary veins, a class of cases in which the œdema may be limited to the region served by the obliterated vein.

As soon as the œdema is appreciable at the malleoli, and before it has gained the root of the limb, there may sometimes be detected, by carrying the finger over the course of the crural vessels, a hard, resisting cord, which can be traced to the ring of the adductor muscles; at the same time, pressure occasions deep-seated pain in the course of the vessels, in the popliteal space, and often in the muscular mass constituting the calf of the leg. Sometimes, the crural portion of the internal saphena vein communicates to the exploring finger the sensation of a knotty cord.

The current of venous blood is then nearly quite obstructed, and upon the dull colored surface of the entire limb, bluish arborescence becomes visible, the indication of an existing tendency to form a collateral circulation. The arborisations, sometimes become hard little cords; and then the circulation is soon obstructed by clots of recent formation. There is observed, at a later period, at several points, little red, or bluish, isolated patches of capillaries, which do not escape the process of coagulation taking place throughout the entire limb.

It is a fact deserving of notice, that the temperature of the affected limb does not seem reduced, the hand, applied to the surface of the affected limb, not detecting any appreciable modification of temperature. The skin continues to present a dull white appearance throughout the entire surface of the limb: it is only at the last, when the affection is near its inevitably fatal issue, that we see the toes, and then the instep take on a diffuse bluish color, the temperature of these parts being then decidedly reduced.

Absence of any modification of temperature, while it excludes the idea of gangrene, also excludes the idea of phlegmasia of the cellular tissue. It is equally unusual to observe any red lines similar to those observed in cases of lymphangitis: the glands are only in exceptional cases the seat of abnormal tumefaction, and in some cases, it is only at the autopsy that we can discover that the deep-lying glands which accompany the vessels are a little enlarged; and on section, we find that they present a slight color. Never since my attention was arrested by this subject, have I observed inflammation of the glands, or peri-glandular cellular tissue.

These facts, then, justify me in rejecting both the existence of lymphangitis and of adenitis in the phlegmasia; while the general rule is a profound modification of the venous system, a modification clinically disclosed by the venous cords, regarding the nature of which, pathological anatomy does not leave us in any doubt.

I have said that the phlegmasia is almost never double from the first onset: when both limbs have been seized simultaneously, the obstacle to the venous circulation is seen first in only one side, and that side is generally the left. Sometimes, however, the venous coagulation may be simultaneously observed in the four extremities, but each occurs in succession. The first case I described to you, is one of those rare cases in which the study of the phlegmasia may be made at one and the same time in all the four extremities.

It likewise, sometimes, happens that the phlegmasia does not make its appearance on one side, till it has entirely disappeared from that which was first attacked.

The average duration of an attack of phlegmasia is about three weeks; that is to say, in a phlegmasia supervening in a recently delivered woman, or in a case of cachexia, at the end of three weeks, the œdema has almost entirely disappeared, the pain has ceased, and the patients have regained the use of the limb. It is necessary, however, to point out the differences in respect of the duration and termination of attacks of phlegmasia which arise according to the different conditions which exist in the individual patient. The œdema hardly ever commences till the tenth day after delivery. It has been sometimes known not to supervene till the end of the third or fourth week; but in these cases, the determining cause is often undue exercise of the limbs, or some other cause capable of keeping up the uterine fluxion, or some pathological state which often exists in the veins of the uterus. Under normal circumstances, about the third week, when there are no peri-uterine complications, there is little ground for fearing phlegmasia, as by that time the general state of the woman is favorably modified; and particularly, if she do not suckle her infant, the *inopexia* has a great tendency to disappear.

The issue of the malady is favorable, when there is no phlebitis properly so called. The œdema then diminishes little by little: the tissues regain their elasticity: the collateral circulation becomes less appreciable: the capillary vessels are less apparent: pain is no longer felt in the calf of the leg and in the course of the vessels. It is also found, that the vessels, superficial and deep, are less tense: they no longer roll under the finger, and, after a longer or shorter period, they regain their normal elasticity, and are again traversed by blood. Sometimes, however, the internal saphena still remains obliterated in a greater or less extent of its course: some nodosities can still be felt in the course of the femoral vein: and it is not till long after the commencement of the malady, that all trace of vascular lesion disappears. At other times, the affected limbs remain œdematous although the malady be no longer in the œdematous stage: the remaining œdema is then due to obliteration of the veins originally the seat of the process of coagulation. This persistence of œdema may continue for several years: every accoucheur has met with cases of this description. In these cases, the circulation is imperfectly re-established in collateral channels, and the cellular tissue is thickened rather than œdematous, as is shown by its having regained its elasticity to a great extent, and by its not retaining the print of the finger when pressed. The least fatigue, however, occasions pain, and induces embarrassment in the circulation of the affected limb.

Pulmonary Embolism.—Van Swieten and Virchow.—Symptoms of Pulmonary Embolism; Extreme Dyspnœa; Apnœa; Thirst for Air; Sudden Death.—Death takes place from Syncope or Asphyxia.—Œdema of the Lungs, Pneumonia, Gangrene of the Lungs, Hydro-Pneumothorax.—Embolism, pulmonary or cardiac, originating in Uterine or Peripheral Phlebitis.

The phlegmasia may be the cause of a most formidable accident—the breaking up in fragments of the obliterating clots. In some cases, the clot is carried on by the current of blood to the heart, and thence to the pulmonary artery. This grave occurrence has been particularly observed in the phlegmasia which follows delivery. The works of Virchow, the memoir of Ball and Charcot,[1] and the researches of Lancereaux have directed attention to it. Venous embolism has, however, been studied in other morbid states: though rare in cachectic cases, it has been observed as a sequel of phlebitis, as is established by the writings of Velpeau, Briquet, and Azam of Bordeaux.

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[1] Ball et Charcot:—Sur la Mort Subite et la Mort Rapide à la suite de l'Obturation de l'Artère Pulmonaire par des Caillots Sanguins, dans les cas de Phlegmatia Alba Dolens et de Phlébite Oblitérante en général. [*Gazette Hebdomadaire de Médecine et de Chirurgie;* 1858.]

When speaking to you of softening of the brain, I referred to arterial embolism depending upon structural change of the heart, or serious lesions of the pulmonary veins. I propose to-day to treat of venous embolism. Its very great frequency in the phlegmasia of lying-in women justifies my going fully into this subject.

Cases of lying-in women dying suddenly two or three weeks after delivery, are known to every physician; and although the dyspnœal symptoms which immediately precede real death are very different from those observed in syncope, there has been a disposition to attribute death to sudden and persistent stoppage of the heart. Those who have read Cullen's admirable chapter on syncope, and recollect that he recognises pulmonary syncope, must have been led to the conclusion, that sudden death preceded by extreme dyspnœa has its seat in the lungs, and must have been thus led to discover clots in the pulmonary artery, which explained to them the cause of death.

Van Swieten has recorded the results of experiments which he performed on dogs: he showed that blood coagulated by acids in the peripheral veins can be transported in the torrent of the circulation in the form of plugs to the pulmonary artery, there to determine certain phenomena which prove suddenly fatal. He says:—"Tentavi similia experimenta in canibus sæpius, vidique semper sanguinem inde grumescere, et per venas semper latiores in suo decursu, ad cor dextrum deferri: dein in pulmones, ibi autem hærebat: et post *summas anxietates*, animalia hæc moriebantur, citius vel serius, prout major minorve talium coagulantium quantitas venis injecta et diversa foret horum efficacia. Poterit ergo talibus causis subito peripneumonia induci."[1]

Dr. Ball rightly remarks, that no better description could be given of that which we now call venous embolism than this description by Van Swieten.

Observe, Gentlemen, that Van Swieten said that death was preceded by very great anxiety—*post summas anxietates*—and if you attentively peruse the works published on this subject during the last few years, you will see that in nearly every case it was noted, that the patients were seized very suddenly with dyspnœa and orthopnœa, and were a prey to the most terrible anxiety; whereas, death by syncope, as you know, is not generally preceded by embarrassed breathing. The patients feel their strength failing almost before they have time to call for help, and die without any apparent struggle. But in cases of embolism, the death-agony testifies to the existence of impeded respiration, the patients thirst for air—*ont soif d'air*—the anxiety which they experience being similar to that observed in cases of rapid asphyxia from any cause, whether from an aneurism bursting into the bronchi, or a stunning stroke of apoplexy.

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[1] Van Swieten:—T. II, p. 654. Aph. 824: Paris, edition, 1771.

The facts adduced by Van Swieten had been forgotten; and to Virchow belongs the honor of having established by numerous works—the first of which appeared in 1847—that clots formed in the peripheral venous system may be carried into the circulation and occasion speedy death by their arrest in the pulmonary artery.

Therefore, when you see a recently delivered woman suddenly seized with symptoms indicating great disturbance of the function of hæmatosis—symptoms the chief of which are pain in the chest, and great respiratory anxiety, you may conclude that possibly there is pulmonary embolism, and look for signs of coagulation in the peripheral veins. In cases of phlegmasia, the search will not be very difficult, a thrombus of the uterine veins is sufficient to produce an embolism. I recollect a young woman affected with peri-uterine phlegmon, in whom digital exploration *per vaginam* (performed perhaps somewhat brusquely) gave rise to all the symptoms of pulmonary embolism.

Sudden death is not always the consequence of pulmonary embolism: death will not take place rapidly unless the seat of the embolism be one of the principal branches of the pulmonary artery. In the cases in which the migratory clot has been able to reach subdivisions of the second or third order, the sudden dyspnœa which is induced may be succeeded by peri-pneumonia and pulmonary œdema, from which the patients may recover. Gangrene may result from pulmonary embolism; and should the gangrene involve one of the peripheral lobules, there is reason to dread a mortal issue, from perforation and consecutive hydro-pneumothorax. The first case of pulmonary embolism which I saw was of this description.

Gentlemen, I am aware that experimentalists have written that large foreign bodies may be introduced into the pulmonary artery of dogs and horses without producing the slightest dyspnœa. I cannot deny the accuracy of this statement, but I may be allowed to say that I think it requires to be tested by new experiments.

I am also aware that it is difficult to understand how obliteration of one of the branches of the pulmonary artery—which is not an artery of nutrition—should determine gangrene of the lung; but if it be borne in mind that, in the experiments of Virchow, pneumonia was a frequent consequence of pulmonary embolism, it may be granted that, as the embolism can produce inflammation, it may likewise produce gangrene.

The embolus according to its size, according to the point in the pulmonary artery at which it has been arrested, and according to the individual peculiarities of the patients, will produce certain symptoms varying in gravity, the mildest being transient dyspnœa and the worst being sudden death. Let me now state how I explain the dyspnœa and sudden death by the migration of a clot to the pulmonary artery. The patients die from a particular variety of asphyxia, to which attention has not been sufficiently directed.

If you forget the etymology of the word *asphyxia*, and only bear in mind the state of which it is the name, you will not see with physiologists, either a complete or an incomplete asphyxia, merely a diminution or suppression of pulmonary hæmatosis. Two things indispensable for the performance of this function—respirable air and blood—that is to say, the oxygenating fluid, and the fluid which requires to be oxygenated. If air be wanting, there is asphyxia, the degree being in accordance with the extent to which the air is deficient: and if a greater or less quantity of blood do not reach the pulmonary vesicles, to be there vivified by the air, a state of asphyxia will supervene from the obstacle to the arrival of blood. Wherever the obliterated clot may be situated, the sudden suppression of function in part of the lung causes anxiety and dyspnœa. Should the obstacle be sufficiently great to prevent at once, or in a few minutes, the arrival of blood in the lungs, rapid asphyxia, and death will occur. The obstacle is seldom sufficiently great to stop so rapidly the arrival of any blood. When the obstacle is situated in one of the principal divisions of the pulmonary artery, there exist on the distal side of the obstruction the conditions favorable to coagulation *a tergo*, so that the pulmonary circulation soon becomes compromised, and at last impossible. In these cases, the asphyxia may be compared, from the effects it produces, to that caused by closing the windpipe or the principal divisions of the bronchial tubes. If to that state, be added the nervous disturbance and the shock felt by the whole system, an explanation is given of the great and rapid disorder caused by a migratory clot.

According to the view I have now set before you, the patients die by the lungs, and from a particular variety of asphyxia. According to other observers, death is the result of syncope. From this point of view, the circulation being obstructed in the pulmonary artery, the right side of the heart soon becomes filled with blood and is, in consequence, unable to contract, while the left side ceases to act from no longer receiving hæmatosed blood, its required excitant. It is sufficient to remark that, in syncope properly so called, death begins at the heart, whereas in embolism, death begins at the lungs. Moreover, the anxious dyspnœa, the craving desire for air, and the purple visage, demonstrate the existence of asphyxia.

When the clots are primarily formed within the heart, it is not unusual to see them prolonged into the pulmonary artery and its principal divisions : in these cases, there is a certain slowness in the progress of the symptoms, and the sudden manifestation of symptoms which occurs at the beginning of the attack in embolism is not observed. On auscultation, it is found that the heart beats feebly, and that its sounds are irregular; and often, there are morbid sounds.

In certain very unusual circumstances, when peripheral clots become arrested in the heart, and are sufficiently large to obstruct the passage of the blood into the lungs, dyspnœal symptoms may show themselves, similar to those occasioned by pulmonary embolism. In fact, the right side of the heart may be considered as the commencement of the pulmonary artery; but in addition to the dyspnœa and anxiety, physical signs, detected by auscultation, will be present from the commencement of the symptoms.

It would be a mistake to deny the presence of pulmonary embolism, simply because the symptoms of that affection had disappeared. Many cases are on record, in which the symptoms have ceased little by little. Dr. Jacquemier has published a case in which recovery took place. In cases of recovery, it is probable that the clot was small, and capable of being absorbed. When the patients die some days after the commencement of the symptoms, pathological anatomy shows, that the transported pulmonary clots, as well as the indigenous clots, may undergo changes tending to softening or cellular organisation. When there is softening of the clot, there is disintegration of the fibrin; and the circulation may become re-established without there being necessarily observed any of the lesions usually caused by capillary embolism. In cases in which the clots become organised, adhesions form between the walls of the vessel and the connective tissue of the clot; these cellular adhesions may themselves disappear after the lapse of some time.

Such, however, is not the usual course of pulmonary embolism: it usually carries off the patients in some hours or days after the commencement of the symptoms.

In the beginning of this lecture, I said that phlegmasia might be the consequence of phlebitis; but hitherto, I have only been speaking to you about spontaneous phlegmasia. I must now, therefore, say something on the subject of phlegmasia consequent upon inflammation of a vein.

It is not necessary that I should give you a description of phlebitis: I am only desirous to show you how inflammation of a uterine vein, or of any pelvic vein, may become the mechanical determining cause of phlegmasia properly so called.

If uterine phlebitis be suppurative, purulent infection is observed: when it is adhesive or obliterative, a barrier is set up against purulent infection, and the obliterative clots may extend even into the trunk of the hypogastric vein.

If you believe—and the fact has already been demonstrated by pathological anatomy—that the obliterative clots ascend to the junction of the hypogastric and common iliac veins, it will be seen that in consequence of the inopexia of cachectic subjects or recently delivered women, a condition which favors a deposit of new fibrinous layers, the head of the hypogastric clot will be sufficiently large to protrude into the common iliac. You then have the mechanical condition of a phlegmasia on that side; for the clot, always increasing in bulk will ultimately obliterate the circulation in the iliac vein; and that coagulation may extend from the external iliac to the femoral vein. It is a remarkable fact that frequently, as soon as there is an obstacle to the venous circulation, fibrin is precipitated, so to speak, into the valvular pouches, giving rise to the knots in the course of the veins, of which I have already spoken.

You can thus understand how an attack of coagulative phlebitis in the true pelvis may give rise to phlegmasia. Velpeau[1] has published cases which strongly support this interpretation of phlegmasia, as a consequence of phlebitis in a neighbouring part.

Whenever you have reason to believe that there is non-suppurative phlebitis of the true pelvis, you may anticipate the occurrence of consecutive phlegmasia. On the other hand, pulmonary embolism, may be observed as a sequel of phlebitis of the true pelvis, without there having been any phlegmasia as an intermediate pathological occurrence: for that to occur, it is sufficient that a fibrinous clot, originally attached to the uterine or hypogastric veins, should become detached. Consequently, when symptoms of pulmonary embolism have been observed after delivery, it is necessary, should no phlegmasia exist in one of the lower extremities, to search in the uterine and hypogastric veins for the point whence the migratory clot has started.

My opinion, then, is, Gentlemen, that painful white œdema, or phlegmasia, cannot be mistaken for any other kind of œdema. I have just reperused the memoir of Dr. Bouillaud, and have pleasure in stating that the work of my colleague is as complete as if it had been written yesterday.[2] It contains eight cases in which phlegmasia was observed in cancerous and tuberculous cachexia, and as a sequel to delivery. In all these cases, obliteration of a vein was observed and described: Bouillaud mentions that there were clots in the veins, obliterative clots, obstructing the venous circulation, and so producing partial dropsies. Bouillaud's work is the basis of all the anatomical researches which have been made in France, since 1823, into the obstruction of veins.

The conditions under which phlegmasia shows itself, its commencement, its progress, and the vascular lesions by which it is accompanied, are all calculated to eliminate sources of error from the diagnosis. When the œdema shows itself, at a time when it is usually limited to the lower limbs, the patient's general state of health has already presented a group of symptoms suggesting that there may be coagulation within a vein. That general state is cachexia, whatever its cause may be. Forewarned of the possible occurrence of venous coagulation, apprised of the coagulation by a feeling of pain in some part of the limb, struck by the rapid development and limited extent of this œdema, the physician will proceed to discover whether, in the course of the superficial and deep veins, there be at any point a hard cord, or the knots which are produced by intra-venous congestion, and deposits of fibrin in the valvular pouches. At these different points, pressure occasions pain, and particularly in those situations where there exist natural obstacles to the venous circulation, as in the muscular masses, and in situations where there is a confluence of several veins in one chief trunk, such as the popliteal, inguinal and axillary regions.

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[1] Velpeau:—Recherches et Observations sur la Phlegmasia Alba Dolens. [*Archives Générales de Médecine*, 1824, *T. VI*, *p.* 220.]

[2] Bouillaud:—Oblitération de Veines et de l'Influence de cette Oblitération sur la formation, des Hydropisies Partielles. [*Archives Générales de Médecine: T. II*, *p.* 188.]

These remarks will enable you to avoid mistakes. In the cases in which the œdema is irrespective of any cachexia, and the consequence of inflamed varicose veins, it is not so considerable as in cases of spontaneous phlegmasia, and a varicose condition of the limb exists which can always be easily traced back to its cause.

I should not think it necessary to insist on this point were it not that varicose phlebitis may give rise to pulmonary embolism. In two cases of varicose inflammation, observed by M. Velpeau and by M. Briquet, the symptoms of pulmonary embolism were produced by a portion of the venous clot getting into the stream of blood.

To determine with precision whether there has been spontaneous coagulation, or coagulation resulting from inflammation, is sometimes difficult, and is never of clinical interest.

I have still to speak of those very limited coagulations, which may become spontaneously developed in the continuity of a vein, and which, from their small size and slight adhesion to an uninflamed vein, may be apt to occasion pulmonary embolism. Perhaps, in such cases, the physician ought, following White's recommendation in respect of purulent infection, to try to interpose a barrier between the clot and the large veins.[1]

I believe that spontaneous coagulations may be developed in the saphena, crural, or any other vein, and remain limited to a very small extent of the vessel. In that case the œdema and pain only exist in those parts of the venous circulation which were served by the obliterated vein. As regards the pathological anatomy of the affection, it may be stated, that we sometimes see fibrinous clots deposited only where there are valves, while cruoric clots are ultimately deposited in the portions of vein situated between two valvular pouches. If the opinion which I am endeavouring to establish has any value, you can understand how important it will be for the physician to recognise the possibility of these partial coagulations existing; for if he cannot prevent their migration, he may be prepared for the occurrence of sudden death from pulmonary embolism.

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[1] WHITE:—Inquiry into the Nature and Cause of that Swelling in one or both of the Lower-extremities, which sometimes happens to Lying-in Women. Warrington: 1784.

Here, Gentlemen, I must make a modification in my statement in respect of the manner in which death takes place in cases of embolism. Generally, the migratory clot reaches the lung, causing dyspnœa and rapid death by asphyxia; but we can understand that in certain exceptional cases, the clot may be arrested in the right auricle or ventricle. Then, in accordance with the predisposition of the patient and the volume of the clot, the phenomena which belong to syncope will be observed; the heart, surprised, so to speak, by the arrival of the migratory clot, will at once cease to beat with regularity and power, and ere long contractions will entirely cease. In these cases, death will take place by syncope, by arrest of the heart: in fact, the prolonged syncope leads to death. Thus, a particular patient who has had repeated fainting fits, may be carried off by syncope, of which the determining cause has been embolism.

A cardiac embolus will then have produced syncope, just as a pulmonary embolus induces dyspnœa and asphyxia.

I look on these latter reflections as purely speculative: I have not observed a single case calculated to convince you of their truth; but a case, communicated to me by my lamented colleague Dr. Thirial, tells in favour of my views regarding syncope caused by embolism. The details are long; but this you will excuse from the interest which attaches to the case:—

"M. X., fifty-six years of age, the head of one of the principal commercial houses of Paris, of nervo-lymphatic temperament, robust, and of a sound constitution, had almost always enjoyed excellent health. During more than thirteen years, I had been his physician, and had only had to treat him for unimportant maladies, such as rheumatic pains, and slight gastric disturbance connected with habitual constipation. He was subject to hæmorrhoids; and had long suffered great inconvenience from a very obstinate eczema podicis.

"On 20th December, 1861, M. X. sent for me. I was told, that for the five or six preceding days, he had begun, without any known cause, to experience a painful condition of the left calf, producing a certain amount of inconvenience in walking or standing. I detected neither redness, swelling, nor hardness in the affected part: the patient was in a feverish state, suffered from pains in the whole muscular system; and one or two points, rather difficult to localise with precision, were more particularly painful on pressure. From the antecedents of the patient, from his having been, as I have said, somewhat subject to arthritic pains, I was led to look on the affection as simple rheumatism.

"I was, however, not altogether free from doubt; and I even entertained a vague apprehension that the patient might be suffering from some obscure, latent lesion of the vascular system. But at the end of a few days, having seen no new symptom to confirm that suspicion, I adopted with confidence the opinion that the affection was rheumatic.

"I consequently ordered rest, different calmative and narcotic applications, and latterly, as these means produced no decided effect, I resorted to a succession of small blisters dusted with morphia.

"This treatment was continued till about the 10th of January. During the whole of that interval, the patient, in the hope of a speedy cure, hardly ever left his bed, and when out of bed for a short time he was careful to place the left limb extended on an arm-chair. After three weeks of this careful management, the pain in the calf seeming to be gone, I recommended M. X. to remain out of bed for a portion of the day, and to walk about in his room so as to prepare himself for resuming in a few days his former habits and occupations.

"Great was my surprise to be sent for on the same day that I gave these directions; and to find very decided œdema extending from the malleoli to the extremities of the toes.

"The appearance of this œdema could leave no uncertainty as to the true diagnosis. After a new examination, I soon detected, about the middle of the calf, a small hard knotty cord, which was about four or five centimeters in extent, and very slightly sensitive. Here, I ought to state, that on both legs there were slight subcutaneous and capillary varicose dilatations. The case was clearly one of very circumscribed phlebitis, the seat of which was a branch of the saphena vein; and it evidently was to this small venous inflammation that the pain in the calf, which I had considered rheumatic, was attributable. It is probable, moreover, that the nodosity was at first very slight, as, notwithstanding repeated explorations, it had escaped notice, both by me and the patient. Then again, the change of position, and particularly the walking, had favored the development of the œdema, which had no doubt been previously kept in abeyance by rest in bed and maintaining the leg in the horizontal position.

"Next day, 11th January, my accomplished colleague, M. Richet, met me in consultation. He detected the existence of a phlebitis of very limited extent, and also an œdematous swelling, which, however, from the left limb having been kept in an elevated position, was less in size than on the previous evening.

"I carefully sought for a cause, general or local, to which I could attribute the affection. In succession, I passed before me in review the habits of life and peculiarities in the health of the patient likely to throw any light on the subject; but after long inquiry, and careful analysis, I found nothing to show even the probable not to say certain character of the cause. My examination was next directed to the heart, and to the origin of the great vessels. I at once observed that there was slight irregularity, and even intermittence in the pulsations of the heart, and in the pulse; but I soon ascertained, that this irregularity was only accidental, and quite transient. I saw that it was due to the mental emotion of the patient, who was very nervous, and was exceedingly disturbed by the examination. I finally satisfied myself that the heart was in a perfectly normal state. I came to the same conclusion in respect of the organs of respiration.

"The treatment prescribed consisted in the employment of topical solvents, particularly mercurial inunction. I specially recommended rest in bed or on the sofa; and advised the affected limb to be kept as much as possible in an elevated position. The tendency to constipation was directed to be kept in abeyance by injections and laxative medicines; and a restorative diet was ordered.

"Under this treatment, which was scrupulously followed for three weeks, the small venous cord gradually disappeared. By February 1st, scarcely any traces of it were left: another small branch of the saphena then became affected, and presented a slight induration a little above the point previously affected, and towards the outside of the calf. M. Richet was again called in. With the exception of difference in situation, this limited phlebitis, that is, the obliteration of the vein, presented exactly the same characters as the preceding obliteration: it consisted in a very limited and very slightly painful, nodosity. It is important to note, that the œdema disappeared at the end of some days, and was not in any degree reproduced. Similar treatment was resumed, with the addition of tonics, particularly quinine in Malaga wine, recourse being had ultimately to alcaline and sulphurous baths. The second attack lasted only a short time: from motives of prudence, however, the patient kept to his bed, or bedroom, for a fortnight.

"At the end of that period, when the patient felt assured that no return of the malady resulted from his leaving his bed and walking about his rooms—not even the slightest appearance of œdema—he began to drive out in a carriage; and soon, he made some excursions on foot. Under the beneficial influence of this new mode of life, the appetite which had been long much impaired, was soon restored. Strength and plumpness speedily returned. Gloomy forebodings gave place to restored cheerfulness and confidence.

"I must mention, however, that notwithstanding progressive restoration, there still remained a certain degree of weakness in the left limb, which was not accompanied by the slightest pain at the points where the vein was obliterated.

"M. X. had, for nearly a month, resumed his ordinary habits of life; and every one was eager to congratulate him upon his recovery, which, long waited for, had come at last with every appearance of secure permanence.

"But, unfortunately, all was not yet terminated. On the 15th March, I had the mortification to be recalled to the patient in consequence of his having had a new relapse. The malady, on this occasion, in place of creeping about its starting point, went ahead at a bound. It thus advanced to about the middle of the inside of the left thigh. There was a small, hard, knotty, and almost indolent cord, of not more than from four to five centimeters in extent, situated in the course of one of the rather superficial veins.

"It was impossible for M. Richet and me not to feel some anxiety regarding this case, seeing that successive relapses had occurred, when apparently there was reason to believe that a cure had been obtained; and seeing also the rapid ascent of the malady, which had quickly mounted so high as to threaten the great venous trunks with phlebitis. These relapses, all of which occurred without any external appreciable morbid manifestation, revealed the existence of a persistent internal cause, whatever it might be; and we could foresee neither when nor where this cause would cease.

"In addition to topical remedies, I prescribed, with the view of preventing the internal coagulations, the use of iodide of potassium and Vichy water, the latter to be taken with meals. Likewise, as it was evident that the habitual constipation of the patient must cause an impediment to the venous circulation, and so favour coagulations, I ordered a dose of castor oil to be administered every two or three days. Rest as before was again enjoined.

"This treatment was most exactly followed. After eight days, the absorption of the intra-venous clot seemed to be already considerably advanced: at this stage, the little nodosity, on account of the slight extent to which it projected, and its slight degree of sensibility, could not be found without a certain amount of attention.

"It was decided that M. X. was to get up on the 23rd March for the first time since his last relapse; but not having found him so well on the morning of that day, I told him that it was necessary to wait. Feeling better, he rose towards evening, sat at table to dinner, and ate with a good appetite. He passed the evening cheerfully with his family.

"At ten o'clock, he went to bed. In making the necessary movement to stretch himself in bed, he was suddenly seized, in the precordial region, with an acute sensation of pain, which only lasted for a short time. He attributed it to nervous spasm; and mentioned it to no one. Two hours later, he awoke. With a view to place, according to custom, a pillow under the affected leg, he stooped, rather abruptly, to take up the pillow which had fallen at his bed-side: that exertion was followed by a momentary return of the pang. At midnight, he awoke with a feeling of general discomfort, accompanied by slight shivering, which gradually subsided in light sleep.

"On the morning of the 24th March, I saw the patient, who then told me with an anxious air of what had occurred since the previous evening. The statement, I confess, seemed to me rather unusual, and led me to apprehend some new incident. I auscultated the heart rapidly, and to a certain extent stealthily, to avoid alarming the patient, who followed all my movements with a troubled expression. I thought I heard, at the base of the heart, a slight morbid sound, the nature of which I could not determine with precision. The pulsations were one hundred in the minute, unequal in strength, and, apparently, a little confused. I was, however, disposed to attribute these disturbances of the circulatory system to the extreme emotion of my patient, who, I perceived, was engrossed with the incidents of the night, and with my examination.

"I prescribed an antispasmodic draught, did my best to reassure him, advised him to rest quietly in bed, specially telling him not to get up in the evening, unless he felt perfectly well.

"From prudential reasons, the patient only took a basin of broth to his breakfast. He passed a good day: in the afternoon, he received some visits. At half-past four, feeling himself comfortable, he resolved to rise, and dine with the family.

"He got out of bed, and sat in an arm-chair to dress himself. He had hardly pulled on his drawers, when he was seized with an indescribable pang at the heart, and felt that he was fainting: he had only time to call his wife to his aid when he became insensible.

"I was sent for in all haste. By a fortunate chance, I arrived at the house at that very moment. I found the patient seated in his arm-chair: he had recovered consciousness, but was icy cold, livid in the face, sunken in the eyes, almost pulseless, and making efforts to vomit. With all possible expedition, I laid him on his bed, placing his head somewhat low, and causing him to swallow a few drops of a cordial: I subsequently had recourse to external and internal stimulants of every kind. For more than an hour, during which time I was a prey to the greatest anxiety, I fought against the syncope, against the state of profound collapse, which lasted so long that I dreaded every moment a fatal issue. Of all my efforts, my greatest was to give courage to the unfortunate man, who believed himself to be hopelessly lost.

"My endeavours were in the end successful; little by little reaction set in, and ultimately became pretty powerful: the pulse rose to 108; but the pulsations were for a long time small and concentrated.

"During the evening, M. Richet met me in consultation on the case. After describing to him all that had occurred since the previous evening, we proceeded together to make a careful inquiry, with a view to discover, if possible, the reason of the serious incidents. Let me here remark, that during the whole of the crisis, I observed neither notable disturbance of the respiratory functions, nor any true dyspnœa.

"Our examination by auscultation and percussion informed us, that there was nothing abnormal in the respiratory functions. It was otherwise in respect of the circulatory system. The pulse, as I have already said, was pretty high being 108, but did not present any notable irregularity. At the base of the heart, Dr. Richet and I heard a slight morbid sound, which recalled the sound I had heard in the morning. This sound, rather difficult to describe, was less like a bellows sound than like a dry clapping. M. Richet thought it proceeded from one of the auricles. That I may omit no fact useful to mention, I may state, that there was considerable gazeous distension of the stomach, which somewhat pressed upon the heart. We on several occasions noticed this state of meteorism, and remarked, that it was not associated with the slightest dyspnœa. Although immediate danger seemed averted, it was evident that the malady was entering upon a new phase, which was one of some gravity and great obscurity.

"Had we had to do with a case of syncope, pure and simple? What was the cause of the syncope? To solve these questions, we reviewed all the probable circumstances, great and small, which could have produced the syncope; such as the state of debility to which the patient had been reduced by his disease and its treatment, the transition from his for some time customary horizontal position, to the upright and sitting attitudes, and the exertion of dressing. But we felt that had the fainting depended on such slight causes, it was not likely to have been either so intense or so protracted.

"We asked ourselves, whether so profound an attack and one of so threatening a character preceded, the previous evening, by functional disturbance of so unusual a character, might not indicate miasmatic syncope?

"And again, might not collapse, so sudden and so prolonged, arise from embolism?

"We considered that we were bound to give prominence to this question, which must evidently raise many objections and difficulties.

"To sum up:—As the syncope might be dependent on debility, or might be the manifestation of a "pernicious" fever, we were decidedly of opinion that quinine was indicated in virtue of its neurosthenic and antiperiodic properties: we consequently prescribed a potion containing 60 centigrammes (9 grains) of quinine and 4 grammes (62 grains) of extract of cinchona: and we likewise advised the patient to abstain from sudden movement and violent exertion.

"25 March.—The night passed without any note-worthy occur rence. I found that he had had some hours of tolerably quiet sleep. In the morning, the pulse was 96; and I noted that the patient had had a natural stool without the assistance of an injection: I use the term *natural*, so as to eliminate from the description anything calculated to cause a suspicion that intestinal hemorrhage was the cause of the syncope. Broth and the quinine potion were ordered to be continued.

"26 March.—After a quiet day, he passed a night even better than the preceding: he had several hours of sound sleep. In the morning, the pulse was 54, and quite regular. There did not remain the slightest morbid sound of the heart. Broth and soup were ordered.

"The family, naturally alarmed by the scene which had occurred two nights previously, expressed a wish that Dr. Bouillaud should join us in consultation. The consultation was fixed for the same evening, when, to our great regret, Dr. Richet was unable to attend.

"Dr. Bouillaud having made himself acquainted with the different phases of the malady, and particularly with the recent crisis, proceeded to the diagnosis with all that care which belongs to him. In the middle of the thigh, he detected a venous cord in process of resolution, and then reduced to a rather thin knotty thread. The respiratory organs were found in a perfectly normal state. He examined the organs of circulation with very great attention. All at once, he detected a certain irregularity, and even a little intermittence in the pulsations of the heart and in the pulse at the wrist: but he soon ascertained that this disturbance was transient, and entirely due to the emotion of the patient, as I had previously observed more than once. Dr. Bouillaud satisfied himself that there was no abnormal sound audible in the heart or great vessels; and he announced that, with the exception of a certain degree of nervousness, there was nothing appreciably wrong with the central organ of circulation.

"I next wished to know Dr. Bouillaud's opinion as to the nature of this crisis which had occurred on the evening before last. I disclosed the uncomfortable impression left on my mind by that spectacle. I mentioned to him my doubts, my different conjectures in relation to that syncope so unusual in its characters; and, in particular, I did not conceal from him my apprehensions of embolism which might or not be well founded.

"Dr. Bouillaud replied that he understood my doubts, and, to a certain extent my fears, suggested by the circumstances of the malady; but, relying upon the very satisfactory result of his examination, upon the good appearance of the patient, and particularly upon the absence of all untoward symptoms since the last crisis, he looked upon the syncope as an accidental occurrence of a purely nervous character. His opinion was, that, in all probability, the case would have a favorable issue.

"Dr. Bouillaud did not hesitate to reassure the family by announcing this conviction, who were only too happy to hear so comforting an opinion from so great an authority. He recommended that the patient should be at once strengthened by tonic medicines and restorative diet; and likewise advised that he should be sent to the country as soon as possible.

"Notwithstanding my great confidence in the enlightened experience of Dr. Bouillaud, notwithstanding the excellent reasons in favor of his prognosis, it was impossible for me to share in his view of the case. The syncope did not seem to me to have been ordinary syncope: certain insidious preliminary phenomena, and the whole group of symptoms, led me to believe that it was of a suspicious nature. Whether it was my solicitude for a patient to whom I was attached by an old and deep affection, or a secret invincible presentiment which possessed me, I know not; but so it was, that I felt that the disease had not yet said its last word, and in spite of myself, I dreaded a new and early attack.

"My apprehensions were so decided, that I deemed it my duty to disclose them to a near relation of the family, so that, while he kept his motives a secret, he might, in advance, point out to the family all the precautions necessary to be taken in the event of a fresh alarm.

"The consultation, however, had produced a happy effect on the patient. Since the last crisis, he had been in a state of extreme anxiety: he had been living under the constant dread of a return of the syncope, which, he said, having come without warning, or any appreciable cause, might any moment unexpectedly return in similar fashion, and carry him off. But the reassuring, convincing opinion of Dr. Bouillaud coming to strengthen my own encouraging expressions, his misgivings were shaken, and he soon even attained to some extent a state of serenity.

"He passed, then, the night of the 27th very quietly. In the morning, the pulse was 80, and perfectly natural. With a return of confidence, there was also a tendency to a return of appetite. During the day, the patient several times took broth and chicken. The cinchona wine was continued at meals. To meet his wishes, the sulphate of quinine, which he had hitherto taken in potion, was prescribed in pills.

"On the 28th March, he had as good a night as on the 27th. He ate with appetite a mutton cutlet at breakfast. As all was going on well: it was agreed, that he should, for the first time since the fainting, be allowed to change his bed, to sit on a chair, and to make his toilet.

"Everybody, particularly the patient, had misgivings as to this trial. I resolved, therefore, to be present, to inspire confidence, and to give succour in the event of its being required. Fortunately, all passed off to a wish, the patient not feeling the slightest discomfort nor the least disturbance of respiration or circulation. During the day, he received some visits with an air of greater satisfaction than he had shown for a long time. He dined on soup and chicken. He passed the evening after dinner with the family. He joined willingly in conversation, and even read aloud the newspaper.

"At ten o'clock, the family retired to rest. At eleven, M. X. was asleep. After sleeping quietly till two in the morning, he awoke. His wife, who lay in an adjoining bed, rose to administer to him his usual cinchona pill. He sat up to take the pill, swallowed it, and then drank a mouthful of water. Having done so, without the least complaint of discomfort or suffering, he again lay down.

"Madame X. had hardly returned to bed, when she heard her husband groaning as if suffocated. She called him, and asked him if he were suffering. He replied in the negative. She lay down at the foot of his bed; and then saw that he was pale, exhausted, unconscious, and motionless.

"I was sent for in haste. As I lived in the same building, I was with my patient in less than ten minutes. What a spectacle! Some hours previously, I had left him full of life and hope: I now found him icy cold with death stamped on his face: he was pulseless, there were no appreciable movements of the heart, and only some respirations at long intervals. In vain I tried to restore him by the use of stimulants. In five or six minutes he expired.

"As there was no autopsy, some degree of uncertainty must remain as to the cause of death. Nevertheless, I do not hesitate to attribute it to embolism: the phases of the malady, the order in which the symptoms occurred, the most characteristic phenomena considered by themselves, all the facts seem to concur in favor of this interpretation.

"It has appeared to me that a complete and faithful picture of the disease could alone supply, up to a certain point, the want of a *post-mortem* examination. This is my reason for giving the complete details. The reader, having been present, as it were, at all the catastrophes of the case, and taking part in all my impressions, doubts, and divergences of opinion, is in a position to judge for himself as to the nature of this affection, which was so irregular, obscure, and insidious in its course.

"Among cases of the same class, this case presents a special, and I may say exceptional, character: for a long time, there existed certain indications leading to the suspicion of embolism, to a certain extent enabling us to foresee, but, unfortunately, not to prevent, the fatal catastrophe.

"In the first phase of the affection, we followed the development of the phlebitis, the expression of a probably general but very obscure cause; and we saw this phlebitis, at intervals of varying duration, give rise to very circumscribed coagulations in the lower extremities.

"So long as the malady, which was then moreover slight, did not go beyond the leg, there was nothing to create much anxiety. But I began to be anxious when the phlebitis went on without interruption, to gain the middle of the thigh, and approach the neighbourhood of the great vessels; for, from that moment, it was impossible to say where the morbid process might terminate, and what might be its consequences.

"The second phase soon made its appearance; and with it came the worst consequences which could have been present: this phase was completed in two periods separated by an interval of only a few days.

"Its commencement was marked by the great crisis of 23rd March: as the sequel of some preliminary symptoms, it burst forth in the form of a frightful fit of syncope, occurring when the patient was getting up for the first time, and just as he was beginning to dress. As the crisis, notwithstanding its violence and duration, had no evil consequences, doubt as to its real nature might, for a moment, be entertained; but the progress of the disease left no room for uncertainty on that point; and it would be difficult, after the fatal issue, not to recognise the effects of embolism in the first seizure.

"To explain the case, I conclude that, from the coagulum seated in the thigh, and in full process of being absorbed, or perhaps from some unobserved coagulum, a small fibrinous or cruoric fragment had been detached, at the moment when the effort was made; and I likewise suppose that this fragment, becoming quickly involved in the current of the circulation, reached the right side of the heart: thence arose, in my opinion, the sudden syncope—the collapse so profound and so threatening.

"The heart, however, under the influence of stimulants became roused, and recommenced its movements. The clot—supposed to be small and soft—taken up, and beaten as it were by the wave of blood—though, perhaps, not dissolved, was broken down into grumous particles sufficiently small to be lost in the ramifications of the pulmonary artery. It has been seen, moreover, that the heart, for some time after the accident continued in an agitated disturbed state: it may be remembered that several hours after that occurrence, I detected an abnormal sound in the heart: it is no doubt quite possible that this sound was simply dynamic, but it is also possible that it had some relation to a fibrinous concretion adherent to one of the orifices or to the columnæ carneæ of one of the cavities of the heart.

"Be the explanation what it may, the greatest crisis, notwithstanding all my fears, had not a disastrous issue. During the two days which followed it, there was a tendency to a restoration of calm: the patient gradually regained confidence, appetite, and good sleep: all the functions were performed in a natural manner: and the state of the heart was quite normal. Nothing presaged immediate nor even remote danger. On the contrary, everything seemed to justify a favorable prognosis, and two days elapsed during which no sign of contrary import manifested itself.

"This calm, however, was a deceitful calm. At a moment when it was least to be expected, in the silence of the night, on awaking from a tranquil sleep, the patient was, all at once, after a slight effort, struck as if by a thunderbolt: he was heard to groan, and in less than an hour he was dead.

"The first seizure was only a menace: the second led up to the fatal issue: but in my opinion, both seizures proceeded from the same cause. It is very probable, that a clot, already free, or only slightly adherent, was shaken about and put in motion by the effort which the patient made to sit up; and that within a very short time, it reached the right auricle.

"This clot, whether from its size, or from some other cause difficult to define, became engaged in one of the cavities of the heart, and immediately determined syncope with profound collapse from which there was no rally."

Gentlemen, if a *post-mortem* examination had been made, it is probable that it would have demonstrated the cause of the syncope to have been in the right side of the heart. The cause was not an organic disease of the heart, for neither general nor local signs of such an affection had ever been discovered. On the other hand, when it is considered, that upon two different occasions, with a long interval of time between them, when making the necessary exertion to dress, he was seized—suddenly, on both occasions—with an indescribable pang at the heart—that he fainted and lost consciousness, is not one led to conclude, that a migratory clot, detached from a vein by the exertion, took the heart by surprise and impeded its action, causing forthwith pain, heart-pang, and mortal syncope?

Pathological Anatomy of Phlegmasia.—Œdema of the Subcutaneous and deep Cellular Tissue of the affected Limbs.—Coagulation of the Blood in the superficial and deep Veins.—Fibrinous and Cruoric Clots.—Fibrinous Clots in the Valvular Pouches.—Absorption of Intra-venous Clots.—Tendency in these Clots to become organised.—Cellular organisation of these Clots, and the Permeability of the New Tissue.—Persistent Fibrous Obstruction of the Veins: Collateral Circulation.—Pseudo-purulent Softening of the Clots.—Organic Causes seemingly Favorable to Intra-Venous Coagulation at particular points.—Absence of Lymphangitis and Adenitis.

Let us now consider the pathological anatomy of phlegmasia alba dolens. In the present day, there are only two views on this subject which merit discussion. In painful œdema, is there a lesion of the lymphatic system? Ought we not rather to believe, that a structural change takes place in the venous trunks and branches?

I have already remarked, that the œdema of phlegmasia shows itself at the extreme and most dependent parts of the body. Thus, it begins in the feet and ankles, then shows itself higher up, while, at the same time, it is chiefly conspicuous in the lower extremities and most depending parts; that is to say, at the posterior part of the calves, thighs, and trunk, when the patient is lying on the back. The skin is tense, the dermic meshes are enlarged; and there are perceived, on the dead as well as on the living body, a blue marbling, arising from a fretted state of the dermis, and its greater transparency in the situations where it is fretted. The limb is deformed and rounded, and presents at the joints an appearance of being strangled.

When incisions are made in œdematous limbs of this description, there pours forth a large quantity of colorless or slightly yellow serosity, in which float numerous globules of fat. Sometimes, the serosity retained in the meshes of the cellular tissue under the aponeuroses and in the intermuscular spaces, seems coagulated, and pits on pressure. The muscles, isolated in the midst of the serosity, do not seem to be penetrated by it; but they are pale, and very flaccid. Vessels and nerves are either dissected, or are in such a state as to be very easily dissected. In situations where the sheaths of the vessels are provided with cellular tissue, the serosity is very abundant.

Deeper incisions show that many veins of variable size are distended by black coagulated blood, or by small yellow fibrinous concretions. If, then, proceeding in our examination, we ascend from the foot to the origin of the affected limb, and dissect the superficial and deep veins, we observe, that the principal trunks are hard, that they roll under the finger, and that sometimes the circum-vascular cellular tissue creaks under the scalpel; but invariably, throughout a greater or less extent, the vessels feel resistant to pressure: their walls seem distended, here and there, knots, corresponding to the valves of the veins, being visible. Sometimes, the walls of the veins retain a great amount of transparency, so that the vessels can be seen to be filled with black coagulated blood. At other times, a vein has a dull appearance; and when laid open by the knife throughout its whole course, it is seen that the clots vary in different places in color and consistence. The blackest clots are generally found at the most distant parts and at the periphery of the limbs; and the nearer the clots are to the trunk, the more fibrinous and yellow do they appear. However, it is not unusual, in the deep veins of the calf and thigh, to meet with very resistant clots in which the fibrinous exceeds the cruoric portion.

The clots in which the fibrinous part predominates are probably the oldest, that is to say, those which first obstructed the circulation. Indeed, the situations in which pain is first felt are those in which the fibrin is found to predominate; as, for example, at the point where the saphena opens into the crural, and in the deep veins of the calf.

There is still greater reason to believe that the cruoric clots, by far the most extensive, are formed consecutively. It is important to remark, that it is at the places where the fibrin predominates, that one can study those modifications which cause certain parts of softened clots to be carried into the current of the circulation to become migratory clots, emboli, the consecutive anatomical phenomena produced by which we shall have to determine.

The fibrin, in certain cases, may have a tendency to become organised: the clot then presents interstitial cellular partitions placed obliquely, transversely, or parallel to the axis of the vessel: some of these partitions or trabeculæ, are inserted in, and incorporated with, the internal coat of the vein. Fibrin, yellow or nearly white, is found in the spaces between the trabeculæ. The organisation of the trabeculæ is not of a doubtful character: in the lamellæ which are most resistant, and evidently oldest, the microscope discloses a fibro-cellular structure, a considerable deposit of amorphous matter, and numerous fat globules: in the lamellæ in course of formation, a similar structure is found, except that the fibre-cells are fewer in number and less distinct. The fibrin within the partitions is granular, and contains some more or less altered blood-globules.

All these anatomical details were verified in a patient treated in my wards, whose case is reported by Dr. Dumontpallier, who, aided by M. Charcot, studied the appearances under the microscope.

We have seen that the obliterative clot consists of two very distinct portions—a cruoric, by far the largest, and a fibrinous portion. The latter is the most interesting to study in relation to its ulterior modifications. One of four things may occur: the clot may break up, and be slowly absorbed—it may burrow parallel with the axis of the vessel—it may hollow out irregular cavities, and present an appearance similar to cavernous tissue—or it may become transformed into a fibrous cord. In the first three cases, the circulation is re-established: in the last mentioned case, it finally ceases. When the clot has undergone the fibrous transformation, it adheres strongly to the walls of the vessel, which at these points is contracted on itself. There is no doubt as to the fibrous character of these clots: and sometimes, they are even infiltrated with calcareous matter, which renders them very resistant. At the points where the clot is thus transformed, it is not unusual to see the *vasa vasorum* of the vein increased in number, and sometimes in diameter.

Every venous obliterative clot may undergo connective transformation; and that transformation may lead to a persistent obliteration of the vessel converting it into a fibrous cord, or producing a cavernous, trabecular, and permeable condition of the vein, through which the circulation is re-established.

These different modes of transformation are the result of a process of organisation seated in the clot, and probably originating in its fibrinous portion. In these cases, the fibrin has an organising power, the last term of which is the production of connective tissue. At other times, the fibrin becomes softened throughout its whole extent, and then nothing is found in the vessels except more or less diffluent masses, of a brown or yellow color, and mingled with perfectly recognisable blood. Again, at other times, these soft masses are encysted, so to speak, in portions of hard clot adherent to the walls of the vessel, and in these cystous cavities, we occasionally meet with a fluid of purulent appearance, which when examined under the microscope is found to be only amorphous granular fibrin, and a quantity, variable (sometimes very large) of white globules, which, by being mistaken for pus-globules, have led to the conclusion that the clot may be transformed into pus. This opinion may appear tenable, particularly when there is thickening of the walls of the vessel, and when they present the traces of phlebitis. But in cases of phlegmasia, suppuration, properly so called, is exceedingly rare; and the truth is, that the purulent appearance of the clot is usually nothing more than a modification of its fibrinous portion.

But although the clot in softening does not give rise to purulent infection, it is frequently the origin of the exceedingly interesting phenomena upon which Virchow has based the theory of venous embolism.

Before I say more on the subject of embolism, I wish to address some words to you upon the clots observed at the valves of the veins, and on the part which the valves play in causing the coagulation of the blood. The formation of the clots is consecutive to the obliteration of an upper part of the venous system, and is induced by the eddy and stasis of the blood when they are formed in the inferior portion of that system. If the iliac or femoral vein be obliterated, the venous blood, prevented from taking its normal course, makes a way for itself through the collateral veins, the result is a temporary impediment to the entire column of blood below the obstruction, an eddy and finally a stasis, particularly at the valves, which are then depressed by the recurrent stream. This last condition is specially favorable to coagulation of the blood, which then takes place under the double influence of the inopexia which has produced the primary coagulation in the iliac or femoral vein, and of the anatomical conditions which the valves present to the too fibrinous blood. When the primary coagulation is not dependent upon an affection of the uterus, the obliterated veins of which have gradually caused an extension of the obliteration to the hypogastric venous system, when, for example, in the subject of tuberculous or cancerous cachexia, the anatomical condition added to the inopexia, is stricture of the veins at the points where they traverse the aponeuroses, this is what takes place in the axillary and popliteal hollows and in the fold of the groin. That the formation of the valvular clots is consecutive to an obliteration of an upper portion of the venous system, is shown by their being fibrous, by their structure being lamellar and much less perfect than that of the clots situated higher up, which are much more intimately adherent to the walls of the vein. This was well seen in an anatomical preparation presented by Dr. Duguet to the *Société de Biologie.*

Pathological Anatomy of Pulmonary Embolism.—Serpent-head appearance of the cardiac extremity of intra-venous coagulation.—Softening of the Head of the Clot.—Its Rupture.—Pulmonary Embolism of Various Dimensions and Forms.—Occupying Infundibulum of Pulmonary Artery.—Generally Arrested at a spur of the Artery.—Obliterating completely or incompletely one of the principal divisions of the artery.—Embolism sometimes continuous with newly formed clots.—Embolism recognisable by its Structure, Valvular Débris, and Special Prolongations.—Embolism of the Principal Divisions of the Pulmonary Artery causing Pneumonia, Gangrene, and consecutive Hydro-pneumothorax.—Embolism occasioning sometimes numerous Pulmonary Abscesses.

Let me lay before you the report by Dr. Dumontpallier of a case which occurred in my wards, which will serve to establish the doctrine of pulmonary embolism.

A young woman, delivered at the Maternity, during October 1858, left that hospital, nine days after her confinement, in a state which seemed very unsatisfactory.

At the beginning of November, she asked to be admitted to the Hôtel Dieu, that she might there have her infant cared for. At the middle of November, she was seized with pain and œdema of the left lower extremity—*phlegmasia alba dolens* characterised by pain, œdema, and a venous cord, extending into the popliteal hollow. The femoral vein contained an extensive and very obvious coagulum.

The venous circulation seemed gradually to become re-established in the left lower limb, and the œdema had almost entirely disappeared, although the venous cord remained: but, suddenly, the patient felt pain in the right iliac fossa and calf of the right leg, which pain soon disappeared, and was not followed by œdema of the right limb.

During the first days of December, she was suffering more from general discomfort than from definite disease; but on the 8th of that month, she was suddenly seized with pains in the right side of the chest, and difficulty of breathing. The inspirations were short and frequent. On auscultation, moist râles were heard: then, a blowing sound and pectoriloquy were detected in the upper and posterior part of the chest: at a later period, in the lower part of the chest, a bellows sound and egophony were heard. The sanguinolent expectoration was not like that seen in acute pneumonia: from the second day of the thoracic symptoms, the sputa resembled those of pulmonary apoplexy: from the fourth day, they had an increasingly gangrenous odour: and on the seventh day, the patient died, respiration having become more and more rapid before death. The pulse ranged between 130 and 140 in the minute. The tongue was dry, and the gums were covered with sordes. There was no diarrhœa. During the last twenty-four hours of life, the body was covered with a profuse cold sweat.

I had enunciated the idea, that the gangrene of the lung might be due to transport of a clot—part of the clot of the phlegmasia—to one of the divisions of the pulmonary artery. In support of that opinion, I called attention to the abrupt manner in which the pulmonary affection. commenced, the frequency of the respiration, the difference of the expectoration from that of pneumonia, and the rapidity with which pulmonary gangrene was manifested in a woman delivered two months previously, and who had had phlegmasia alba dolens. My attention had been directed to this subject, by the researches of Virchow, and a recently published memoir of Dr. Charcot.

Let me now give you an account of the appearances observed at the autopsy. The crural, saphena, and popliteal veins, and the veins of the calf, were full of colored clots of varying consistence and structure, which throughout the greater part of their extent were free, and only adhered at some points to the walls of the veins.

The femoral vein in its upper part, at the crural arch, contained a fibrinous clot from four to five centimeters in length, of a pink color, perfectly organised, hard to the touch, composed of longitudinal striæ, which in its whole extent adhered to the walls of the vessel. The internal surface of the vein had no downy smoothness, but was so intimately united to the clot by lamellæ and filaments of connective tissue, that it could not be removed without tearing this tissue. The cellular tissue surrounding the vein was indurated, œdematous, and creaked when cut.

The femoral clot was continuous inferiorly with a fibrinous clot mixed with much of the red portion of the blood; it was brown, and this color was of a deeper shade the nearer it was to the popliteal vein. The same femoral clot was continuous superiorly with a semi-fibrinous, semi-sanguineous clot, which was highly organised, but non-adherent, in the common iliac and external iliac veins and in the vena cava inferior.

There was a fibrinous clot in the vena cava inferior, to a certain extent obstructing that vessel. This clot was of a pink color, fibrinous, resistent on pressure, composed of longitudinal striæ, and non-adherent. Its length was five centimeters, and its diameter one centimeter: it was flattened from before backwards, and terminated a little below the mouths of the emulgent veins, in the form of a soft, slashed stump, to which were attached five or six small pedicles of fibrinous clot resembling portions of a lumbricus: some clots of similar nature and form appeared to be unattached. It is well to remark, that the autopsy was performed with every possible precaution, the dissection being made whilst the parts were *in sitû* and in their normal relations. The clot in the vena cava inferior terminated, as I have said, in the form of a stump, and was continuous posteriorly with an exceedingly small membranous fibrinous clot, which was joined to a large fibrinous clot above the emulgent veins. This latter, which was non-adherent, occupied almost the entire cavity of the vena cava, in its hepatic portion, where it received many other fibrinous clots of different sizes belonging to the sub-hepatic veins. It reached the right auricle, and then extended into the ventricle, after sending off a fibrinous prolongation into the vena cava superior and vena innominata. Within the heart, the clot was yellow, fibrinous, and seemingly adherent, from its numerous prolongations extending into the interstices of the columnæ carneæ.

The entirely fibrinous cardiac clot was continuous with a clot, partly fibrinous and partly cruoric, situated in the pulmonary artery and its two principal divisions, extending likewise into divisions of the second and third order of that vessel. These semi-fibrinous, semi-cruoric clots had evidently been formed during the latter moments of life and after death, judging from their structure and slight consistence. I shall afterwards have to speak of other clots, some of which were found in the principal divisions of the pulmonary artery. At present, I proceed to describe the anatomico-pathological examination of the parenchyma of the lungs, more particularly of the right lung. The entire examination, I repeat, was made with the organs remaining in their natural situation.

The right pleura contained a sero-purulent effusion, and purulent false membranes: the lobes of the right lung adhered to one another by cellular adhesions and false membranes.

The surface of the inferior posterior portion of the upper lobe of the right lung had a dark brown color for about from four to five centimeters square. In that situation, the pulmonary tissue was exceedingly soft; and, on insufflation, it was found that the lung was perforated at the place where it was gangrenous. The perforation had not taken place till the last moments of life; or perhaps, not even till after death; for the patient never had any signs of pneumothorax.

The perforation of the lung led to a gangrenous anfractuosity, large enough to contain a pigeon's egg. The appearance of the pulmonary tissue, and the odour of the affected parts, left no doubt as to the gangrenous character of the local lesion.

That having been ascertained, I resumed the dissection of the pulmonary artery. I observed that the large branch which serves the superior lobe of the lung contained a fibrinous clot adherent to the walls of the vessel: it was of a pink color, had very well marked longitudinal fibres, and was in all respects similar in structure and appearance to the clot found in the vena cava inferior. It was three centimeters in length, and was posteriorly continuous with a much less organised fibrinous clot, and continuous anteriorly—that is to say, towards the divisions of the third and fourth order—with softened cruoric clots.

The principal clot of the pulmonary artery was not in any degree softened, and was, in all parts, of uniform consistence.

The portion of lung which presented signs of hepatisation and gangrene corresponded with the division of the pulmonary artery obliterated by the fibrinous clot.

I am not going to endeavour to explain how obliteration of a division of the pulmonary artery may occasion gangrene of the lung: I only wish to remind you that Virchow, by his experiments on dogs, was led to believe this possible.

I must add, that in the case now under consideration, I found no obliteration of the bronchial arteries and veins to account for the gangrene of the lungs. I found obliteration of the pulmonary artery coexisting with gangrene of the pulmonary tissue. It is probable, that the embolus which caused the obliteration also caused the gangrene.

There was no lesion of the left lung.

The examination of the uterus and its appendages presented nothing requiring mention. There were no traces of inflammation in the utero-ovarian and hypogastric veins.

It may be concluded, from the facts now stated, that the phlegmasia alba dolens was due to the obliteration of the crural vein; that the crural clot was prolonged, by juxtaposition of fibrin, into the vena cava inferior; that the fibrinous clots (both those which were free and those which were adherent to the surface of the clot of the vena cava) might have given rise to venous embolism; that at the time when chest symptoms supervened, an embolus was carried into the pulmonary artery, and arrested in the superior division of that vessel in the right lung; and that gangrene was the result of this obstacle to the pulmonary circulation.

The fibrinous coagulation met with in the other divisions of the pulmonary artery, vena cava superior, heart, and hepatic portion of the vena cava inferior, seemed to have been produced progressively by the obstacle to the pulmonary circulation during the latter hours of life.

To sum up:—A recently delivered young woman was affected with phlegmasia alba dolens; and three weeks after the commencement of that malady, when she believed that she was cured, she all at once felt acute pain in the right side of the chest. Very soon, the expectoration of pulmonary apoplexy was observed, which expectoration afterwards acquired a gangrenous odour—and the patient died. The lung was found in a gangrenous state, in that portion served by a branch of the pulmonary artery obliterated by an old fibrinous clot, similar in all respects to another peripheric clot found in the vena cava inferior, which at its free portion had a jagged appearance and marks of being torn. It is reasonable to suppose that during life, probably under the influence of an exertion, and of a strong wave coming from the emulgent veins, a portion of the clot of the vena cava, softened at its superior extremity was broken up, detached, and then carried to the lung, where it determined the symptoms which have been described. This migratory clot, by becoming arrested in the pulmonary artery, first occasioned a hæmorrhage, and then gangrene limited to that portion of the lung to which the obliterated branches of the vessel were distributed.

Now that you are acquainted with the conditions in which embolism may occur, and the phenomena to which it may give rise, I proceed to describe that anatomical state of the clot which favors the formation of venous emboli.

All observers agree, that the superior extremity of the venous clot has a special form. The venous clot has a tendency to become prolonged towards the heart, and generally goes on extending till it reaches the mouth of some considerable branch. That you may quite understand this disposition, let us suppose a clot spontaneously formed in the crural vein: it has been observed that such a clot will generally become prolonged into the external iliac to the mouth of the hypogastric vein: there, new layers of fibrin, furnished by the hypogastric circulation, will give particular form to the clot. In that situation, the superior extremity of the clot often takes the shape of a slightly flattened serpent's head, free from adhesions and continuous with the original clot.

The head of the clot is formed of two parts, viz. the cortical, which is resistent and fibrinous, and the central which is softened: it is to this central softening, that the head of the clot owes its flattened form. The softening also leads to other and much more important consequences: it first of all extends to the peripheral parts of the clot, and there induces disintegration, so that it becomes easily torn: then, the portion of the clot set free in this way may be carried by the circulation and give rise to the phenomena of cardiac or pulmonary embolism.

In fact, the head of the clot constantly receives the shock of the blood carried by the nearest afferent vein; and if the clot be softened at any point, we can understand that a portion may become detached, and be carried on to the vena cava, the heart, and the lung. It will there produce symptoms exactly similar to those which Virchow produced at pleasure, in his experiments, by introducing foreign bodies into the jugular vein. The portion of clot liberated from its attachment, in the manner I have described, is simply a foreign body, which will advance towards the heart till stopped at the angle formed by a branch of the pulmonary vein, or by some subdivision of that vessel too narrow for its further passage.

The remaining portion of the peripheric clot then presents a slashed fibrinous and cruoric extremity, floating like the fibres of the root of a tree; or, it may be entwined, so to speak, in new fibrinous clots—clots formed in the last struggle of life—which may, as in the case I described, become so prolonged, as to extend with apparent continuity from the original clot to the pulmonary embolus.

This apparent continuity of clot has furnished the opponents of the theory of embolism with an objection. They say that in the case the details of which I have now laid before you, and in other similar cases, what is seen is only the continuance and extension of the femoral clot into the vena cava inferior, right auricle, and pulmonary artery. In reply to this objection, it is sufficient to state, that the structure of the clots is quite different. Though the structure was the same, the clot in the vena cava inferior was of as old a date as that in the pulmonary artery: it was quite otherwise in the space intervening between these two extreme clots: this was a fact not admitting of any dispute, as the interposed clot was fibrinous, gorged with serosity, a veritable fibrinous sponge similar to the clots observed in the right side of the heart consequent upon the final struggle. The clots situated at the extremes, although formed of fibrin were resistent, the fibrin having undergone retrogressive changes. Moreover, it has been ascertained by clinical observation, that there had been œdema of the left lower extremity at a particular period, suddenly followed by urgent dyspnœa, and pathological phenomena attributable to the lungs. Bear in mind this important fact, that in our patient, the contractions of the heart had regained their natural frequency and amplitude: it was not till after the lapse of some time, that all terminated in the pulse becoming small and irregular and the breathing exceedingly disturbed.

It being admitted, that there has been an interval of variable duration between the phenomena attributable to pulmonary embolism and to death: it being, moreover, admitted, that the intermediate clot was of most recent date, the opponents of the theory of embolism object, that the old pulmonary clot might have been indigenous—that is to say, that spontaneous coagulation had taken place in the pulmonary artery, just as it might have occurred in a peripheric vein, the crural vein, or any other vein. To this objection, clinical observation replies forthwith, that the thoracic phenomena have not occurred till a period long after the existence of the phlegmasia; that the commencement of these thoracic phenomena did not take place till the phlegmasia had been in existence for some time; that the first manifestation of the phenomena supervened with, and was accompanied by, sudden maximum dyspnœa, and that it is impossible to say how rapidly there might be formed in one of the branches of the pulmonary artery, an obliterative clot capable of originating formidable dyspnœal phenomena, or even sudden death. Finally, pathological anatomy has shown, that the clots which produce embolism have a special form and structure, sometimes presenting distinct, indubitable, marks of their peripheric origin.

These clots, generally stopped at a spur of the pulmonary artery, are only adherent by one part of their surface to the vessel; they are split into two parts, of which the central is soft, and the peripheral a fibrinous envelope. They sometimes retain the form of the serpent's head belonging to the peripheric clot, and also the jagged extremity which united it to the latter. The clot, become an embolus, is continuous, above and below, with new fibrinous and cruoric clots. When we recollect, that in cases of spontaneous intra-vascular coagulation, the blood has a special constitution, we can easily understand how the embolus should become a centre around which a new fibrinous formation is formed. It often happens, that the embolus is lost in the midst of these new clots, and that some trouble is necessary to find it.

So much for the embolus, its relations, and its continuity with secondary clots: its structure is identical with that of the peripheral clot: the solvent power of ether, and, still more, the solvent power of sulphuret of carbon, show, that the embolus and the peripheric fibrinous clots have the same composition; which renders it exceedingly probable, that the embolus has been detached from one of the clots.

For the sake of those who may be but little inclined to accord conclusive value to the arguments I have now adduced, let me add, that there are emboli which bear indubitably the stamp of their origin: they present marks which leave no doubt as to the place whence they came. Dr. Lancereaux, in a communication to the Société de Biologie, has stated, that he has seen a clot bearing the print of a venous valve. It has likewise been stated, that some clots have been found to have carried with them valvular debris discoverable in the substance of the clot. I am indebted to Dr. B. Ball for the account of a very remarkable case of pulmonary embolism, in which the clot presented three lateral prolongations. After minute and protracted research, the starting point of the migratory clot was found in the vena azygos. At the upper part of that vessel, there had been rupture of a clot, and immediately above that rupture, the vessel presented three lateral orifices, which, in situation and calibre, perfectly corresponded with the disposition of the three lateral prolongations of the migratory clot.

It is unnecessary to say, that such facts give powerful support to the theory of embolism: they, in fact, are demonstrations of its truth.

Emboli may be capillary, and numerous: that is to say—very minute fragments of disintegrated fibrin may be carried by the circulating stream, and deposited in the capillary ramifications of the pulmonary artery. In these cases, there are observed, on the surface of the lung, numerous small ecchymoses, identical with those which M. Sedillot has described as occurring in certain forms of metastatic abscesses in cases of purulent infection.[1] In the centre of these ecchymoses, the fibrinous nucleus can sometimes be detected: at other times, after remaining for a long period in the capillary branches of the pulmonary artery, these minute deposits of fibrin become softened, and assume a purulent appearance. Some of the ultimate ramifications of the pulmonary artery may become injected with fibrin; and this network of fibrinous deposit may occasion lobular pneumonia. I ought, however, to remark, with Virchow, that these forms of embolus are observed chiefly in the capillaries of the liver, spleen, and kidneys, and consequently originate in structural changes of the ventricles of the heart, and in the large arteries. This fact has been established by the publications of Virchow, and of MM. Charcot and Lancereaux on ulcerous endocarditis.[2]

Let us now consider together, what bcomes of the migratory clots, and what changes they undergo when arrested in the pulmonary artery.

If they are of large size, like those observed in the cases described by M. Briquet and M. Velpeau (in which the immediate result was rapid death), they do not undergo any modification of texture. They only recall, by their form and aspect, the vein from which they came.

Though the migratory clots are less considerable, they may reach branches of the second, third, fourth, and even fifth order of the pulmonary artery, remain there for a certain time, and acquire from their new relations a condition of existence and new modifications.

Though Virchow and Cohn[3] were the first to study these modifications, I have pleasure in here mentioning the published researches of MM. Charcot, Laucereaux, and B. Ball on this subject. They have all asked the same questions. What, say they, becomes of these clots? What changes do they undergo in the pulmonary artery? What is their action on the pulmonary artery, and parenchymæ of the lungs?

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[1] Sedillot:—De l'Infection Purulente, ou Pyohémie. p. 476. Paris, 1849.

[2] Comptes Rendus des Séances et Mémoires de la Société de Biologie: 1862.

[3] Cohn: Klinik der Embolichen Gefüsskrankheiten. Berlin: 1860.

The changes which the clots may undergo in the pulmonary artery are of infinite variety, and may infinitely vary with each case. One may, nevertheless, describe in general terms their relations to the artery and their ultimate modifications.

Emboli vary in size according to the situation in which they have originated; and they are arrested in the pulmonary artery, either on one of its spurs, or in its divisions of the second, third, and fourth order.

The modifications which emboli undergo in the pulmonary artery are proportionate to the period they have been there. Generally adherent by a greater or less extent of surface to the interior of the vessel, they are continuous with the fibrinous or cruoric clots: they are often covered, to a greater or less extent, by newly formed clots; but they can be easily recognised by the modifications which they have previously undergone; thus, their central position is softened, and is composed of a pseudo-purulent yellowish red substance, containing amorphous fibrin and the rudiments of connective tissue, while their extremities are rounded or jagged.

This form and structure, identical with the form and structure of the clots of the peripheric venous system, enable us to recognise their origin. When once the clots are definitively arrested in the pulmonary artery, they there undergo new modifications. The fewer their number, the greater is the chance of their softening. Should they, from their size, become fixed in divisions of the second, third, and fourth order, they may be detected, after a sojourn of longer or shorter duration, either to have undergone no important change of structure, or to have formed adhesions with the pulmonary artery. At other times, like a real foreign body, they determine morbid action, the result of which, according to Cohn and Lancereaux, is the production of a membrane which will form a cup, and then an envelope for the migratory clot, "in such a manner," says M. Lancereaux, "that after no long time, the arterial coagulum is completely surrounded by a perfectly organised membrane." I confess that I have not been so fortunate as M. Lancereaux, having never found capillaries in the clot nor in its membraneous sheath; nor have I ever met with that new membrane to which Lancereaux attributes special properties in relation to the absorption of the embolic clot. I must here, however, recall to your recollection the fact that Virchow, in his memoir on embolism, after describing the connective organisation of the pulmonary clot, has demonstrated the existence of a membraneous sheath around triangular fragments of caoutchouc introduced into the branches of the pulmonary artery. He recognised the existence of capillary vessels in this membrane; and remarked, that the maximum of vascularity corresponded to each of the angles by which the caoutchouc came in contact with the serous coat of the vessel: he likewise pointed out, that there was an unusual development of the *vasa vasorum* at these points. Virchow, also, says that he has observed the formation of vessels at the extremities of the clots, when, apparently there was no continuity between those vessels and the *vasa vasorum,* a circumstance which leads us to infer that a vascularising power resides in the substance of the clots. If it be so, the organisation of a thrombus or an embolus may be independent of the vascular walls.

We see, then, that an embolus may become organised. What aftewards becomes of it? Probably, it undergoes connective, cellular, pseudo-membranous transformation; and, after having been rarefied, may disappear, in the same way that the new membranes of pleurisy disappear. Probably, the complete disappearance of the clot is usually due to absorption, and this absorption can only be effected by the new vessels continuous with the *vasa vasorum.* Here, I ought to state, that progressive disappearance of the clot is often observed during life; and that at the autopsy performed at an epoch very near to that at which the obliteration was noticed, it has happened that no trace of the obliterative clot could be found—no abnormal development of *vasa vasorum,* and no change in the serous coat of the vein. There is, consequently, reason to conclude, that the disintegrated fibrin had returned directly into the circulation.

We have still to study the possible action of the embolus on the pulmonary artery, and on the parenchyma of the lungs. I have already spoken at some length regarding the dyspnœa and extreme anxiety of the patients; and I have stated that rapid death, from syncope, occurs in some cases. The first effects produced by the embolus arrested in the infundibulum, in the trunk of the pulmonary artery, or in one of its principal divisions, are embarrassment of the cardiac circulation, and temporary or permanent arrest of the contractions of the heart. In these cases, cardiac syncope is the peril to be dreaded: but though sudden death, or to speak more correctly, rapid death, has been observed several times, it has generally happened that the dyspnœa diminished some seconds or some minutes after the clot reached the pulmonary artery. In such cases, the heart which had not ceased to beat, gradually regains its regularity and power: it is the lungs which undergo secondary changes in function and structure.

The organic matter, and the fibrin of the emboli, may have a threefold action—an *obstructive* mechanical action, an *irritant* action as a foreign body, and a *special* action (perhaps putrid) which alters the tissues with which it comes in contact.

It appears from the researches of Virchow, Cohn, Charcot, B. Ball, and Lancereaux, that the most usual lesion is œdema of the lung, the direct consequence of the mechanical obstruction; then, next in frequency, comes hypostatic pneumonia, the result of irritant action; sometimes, though seldom, apoplectic extravasated clots; and still more rarely gangrene of the lung.

It is important to bear in mind, that the special nature of the obliterating body may exert an equally special action on the lung: this fact is established by the experiments of Virchow, and by *post-mortem* examinations such as some of those which I have described to you. Such cases, however, are of rare occurrence: Dr. B. Ball only records two which can be placed alongside of my case. I am inclined to think that cases of gangrene from pulmonary obstruction are most apt to occur when there has been morbid alteration of the humors, peripheric gangrenous affection, or putrid infection, whatever may have been the original cause. In such cases, the obliterating clot possesses special properties.

The details into which I have entered, in relation to pulmonary embolism have led me far away from my starting point—phlegmasia alba dolens. But I could not pass over in silence so frequent a complication of phlegmasia alba dolens, a complication the result of which, though only very recently explored, has furnished a great amount of instruction.

Let me now terminate my lectures on phlegmasia alba dolens, by stating certain conclusions which may be considered as a summary of my remarks on that subject.

Obstruction of a vein is the cause of phlegmasia alba dolens. This obstruction is generally the result of spontaneous coagulation of the fibrin of the blood, and sometimes, but much more seldom, of coagulative phlebitis. Spontaneous coagulation is specially met with in patients under cachectic influence and in lying-in women.

The obliterative clots, contain a great quantity of thready molecular fibrin, red or white globules, fat, and hæmatin. These clots becoming disintegrated may disappear without leaving any traces of their past presence, and generally without causing appreciable phenomena. They may undergo, *in sitú*, a process of cellular organisation: the interior of the vessel may assume the appearance of a cavernous trabecular tissue, and the circulation may be carried on by these altered vessels, or the connective organisation may become fibrous, may be accompanied by contraction of the walls of the vessel, which may be transformed into a fibrous or fibro-calcareous cord.

Venous clots have two extremities, the peripheric and the cardiac: the former ramifies in the origin of the veins: the latter often has a resemblance to a serpent's head, and is frequently cystic, in which case, the envelope of the cyst is composed of stratified fibrin, and in the interior of the pouch, there is found softened, disintegrated fibrin, having the appearance of pus. The head of the clots is usually non-adherent, and is situated at the mouth of an afferent vein.

Supported by a more or less resistent pedicle, this extremity may be carried away by the circulatory torrent, arrested in one of the branches of the pulmonary artery, and give rise to phenomena of pulmonary embolism. The secondary phenomena vary with the general state of the patient, and with the extent, volume, and composition of the emboli.

Cardiac and pulmonary emboli may occasion sudden death. Sudden death is the immediate result of the arrival of the embolus in the heart, or in the trunk or principal branches of the pulmonary artery. The embolus may become fixed in the divisions of the pulmonary artery, and there undergo various changes. The clot may be absorbed at the place where it was disintegrated, or it may obliterate the canal of the vessel. Persistant obliteration will generally produce local anæmia, œdema, and pneumonia—less frequently, gangrene of the lung and pneumothorax.

LECTURE XCVI.

PERINEPHRIC ABSCESS.

Insidious Beginning and Slow Progress of Perinephric Inflammation.—Etiology of Perinephritis: Fatigue, Muscular Exertions, Contusions, Repeated Blows over the Kidney.—Renal Calculus.—Typhoid, Purulent, and Puerperal Fevers.—Perinephritis causing Sympathetic Pain in the Bladder and Spermatic Cord.—Perinephric Abscess Consecutive to Iliac Abscess, Typhilitis, and Hepatic Colic.—General Symptoms.—Local Symptoms.—Intra-abdominal Tumour in the Side.—Iliac Abscess.—Spontaneous Opening of the Abscess into the Lumbar Region, the Intestine, Bladder, Vagina, and (very rarely) into the Peritoneum.—Lumbar Fistulæ.—Relative Gravity of Perinephritic Abscesses.—Treatment: Opening by bistoury in the Iliac and Lumbar Regions.

Gentlemen:—You may, for many months, assiduously follow an active clinical service, without having a single opportunity of observing a case of perinephric abscess. For this, there are two reasons: the first is, because the affection is relatively rare; and the second is, because the nature of these cases is liable to escape notice, attention being only directed to the concomitant and consecutive phenomena. The beginning of the disease is often as insiduous, as its progress is sometimes slow: for a considerable time, the local symptoms may be absent; and sometimes, the general symptoms are so predominant, that little attention is paid to the patient's complaint of pain in the side. When the pain cannot be attributed to lesion of the stomach, liver, lung, pleura, uterus, or annexes of the uterus, there is reason to suspect that the tissue which envelopes the kidney is diseased, particularly when nothing morbid can be detected in the urine. In the beginning of the affection, the patients may present such lesions in the iliac fossæ and broad ligaments that there seems good ground for ascribing all the morbid phenomena to the lesions.

There is sometimes a very great difficulty in recognising perinephric abscess; and this difficulty continues, till the local symptoms become so marked as to render it impossible not to suspect the cause of the malady. Analyses of the different cases which I am about to lay before you will prove that this is a real difficulty, and put you on your guard against chances of error.

A woman, 32 years of age, was the occupant of bed No. 2 Saint-Bernard's ward. For ten days, she had been complaining of pains in the kidneys, and of feverish symptoms. Every afternoon, from the day on which she began to experience these pains, she was seized with fever and shivering: the fever lasted several hours on each occasion, and prevented the woman from going to sleep till one or two o'clock in the morning. During the continuance of the fever, she had lancinating pains in the right side.

At the middle of June, that is to say, five days after her admission to the Hôtel Dieu, the fever became continuous with paroxysms which returned every evening from four to five. The paroxysms frequently began with a great shivering fit, and sometimes there was slight shivering in the afternoon.

There was almost total want of appetite, great thirst, nausea, and vomiting. She was rapidly getting thinner, and there was prostration of strength without any stupor. There was neither diarrhœa, nor pink lenticular spots. On several occasions, purgatives were prescribed. Three weeks after admission to the hospital, the patient had regained her appetite, and was decidedly improved. Twenty-eight days later, however—on the 10th July—having been exposed to cold, she had a return of the fever, accompanied by rigors, and it was stronger than ever: new and very acute pains were complained of in the abdomen, on the right side; while, at the same time, there was flexion of the thigh on the pelvis. Soon afterwards, a very decided swelling declared itself in the lumbar region: the costo-iliac hollow was effaced.

On grasping the loins with both hands, and exerting slight pressure, one could distinctly feel that there was deep-seated boggy distension in that region. Day by day, the local pain became more acute on pressure: it lancinated several times during the day; and in the afternoon, the patient had rigors followed by fever.

The fever was continuous, and the rigors recurred at intervals: the pain became more and more acute in the lumbar region, where it could hardly be doubted that pus was forming. Some days later, fluctuation having become evident, M. Jobert (de Lamballe) afforded an exit to the pus by making an incision, some centimeters in length, in the lumbar region. Before penetrating into the purulent collection, the bistoury cut through a thick layer of œdematous indurated tissues. Two small lumbar arteries were tied. The pus, which issued copiously from the incision, was greenish white, and contained streaks of black blood. To stop the flow of blood from the wound, small strips of agaric were introduced between its lips. Before making the dressing, I introduced the index finger into the abscess, and found that the kidney was pushed forwards, and that the cavity of the abscess contained debris of cellular tissue adherent to the surface of the kidney.

Immediately after the operation, the patient felt relief: she had three hours of sleep during the day. In the evening, the strips of agaric were removed to afford issue for the pus. The pus still contained small filiform clots. The lips of the wound had ceased to bleed. A pledget of lint was introduced into the abscess.

Next day, the patient was almost without fever: the pulse was rather frequent, but it was quite regular. The patient took a basin of broth with relish. Day by day, the febrile state decreased: the walls of the abscess closed on themselves, and the purulent discharge went on diminishing. The patient ate a ration of food; and made visible progress towards recovery. At last, the fever quite ceased: the appetite improved: a small quantity of purulent serosity, devoid of fœtor, exuded from the incision. The distended boggy feel of the abscess no longer existed. Three weeks after the operation, the incision was cicatrised. Some months after leaving the hospital, the patient returned to see us: she was then quite well, and had regained a certain degree of plumpness. She could walk easily, and without being tired. From the time of her leaving the hospital, she had not felt any pain in the lumbar region.

This case, Gentlemen, may seem to you simplicity itself; and in now hearing my rapid sketch of it, you may possibly be surprised that after the complaints latterly made by the patient, the least hesitation should have remained as to the diagnosis of the disease. Those of you, however, who have had some clinical experience, and have listened attentively to the history I have laid before you, will have grasped all the importance of the case. For ten days, this woman had been suffering from pains in the loins, and from a general feverish state: she had been having fever and rigors every afternoon, accompanied (during the febrile paroxysm) by shooting pains in the right side. Along with the fever, she had nausea and loss of appetite. Her strength soon failed so much that she applied for admission to the hospital. When I interrogated her for the first time, she mentioned, in addition to the circumstances which I have now described, that the jolting of the carriage which conveyed her to the hospital had occasioned pains in the abdomen, particularly in the hypogastrium. I found that she had been having every afternoon a febrile paroxysm and rigors.

Any one not taking into account the lancinating pain in the right side, along with the general symptoms, and the hypogastric pain, might have supposed that the case was an incipient attack of mild continued fever; that the hypogastric pains excited by the jolting of the carriage arose from congestion of the uterus and its appendages, a condition often observed at the beginning of fevers. This supposition seemed the more probable, that after some days of expectant treatment, the feverish state perceptibly diminished, the rigors and paroxysm of fever did not recur; and there was, moreover, a return of appetite.

But this was only a pause in the progress of the disease. After exposure to a chill, the patient had a renewed attack of fever, attended by pain in the right side: there soon appeared tumefaction of the right lumbar region, where boggy distension became manifest: the rigors recurred daily: there then supervened a difficulty, and subsequently, an impossibility to stretch the thigh, which from that time became flexed on the pelvis. Henceforth, no diagnostic doubt remained: it was evident that there was an abscess in the renal region; and that the psoas muscle was involved in the inflammatory process. I do not insist upon the termination in this case: I shall afterwards have occasion to revert to the quantity and nature of the pus of perinephric abscesses, and to all the symptoms which follow their being opened. At present, I only wish to impress on you the fact that the beginning of a perinephric abscess is often insidious: attention is not always sufficiently directed to the lumbar pain which is apt to be neglected in consequence of a greater pain existing in another part of the body, and the general symptoms being sufficient to divert observation from the local pain. I beg you also to remark, that in this case the formation of pus took place slowly, and so to speak, at two periods.

In the first case, I was unable to discover the cause of the phlegmasia; and I have told you that the abscess was *primary*, to distinguish it from perinephric abscesses consequent upon a lesion of the genito-urinary organs, or a serious general derangement of the economy.

Before discussing the various causes of perinephric abscesses, I must rapidly sketch the anatomy of the region in which they are developed, and the relations of the kidneys to neighbouring organs. The kidneys are situated on each side of the vertebral column, and surrounded by a large quantity of cellulo-adipose tissue. The fatty capsule of the kidney has relations posteriorly with the pillars of the diaphragm, and with the deep fold of transverse aponeurosis. It has, also, relations anteriorly with the ascending or descending colon. It is unnecessary to describe the connexions of the kidneys with the liver or spleen. The fatty capsule is continuous with layers of cellular tissue, which are continuous with the cellular tissue of all the organs of the perinephric regions. But the continuity most important to note is that which exists between the perinephric cellular tissue of the iliac fossæ. The iliac aponeurosis, to which M. Cloquet has given the name of *fascia iliaca*, is usually only formed, in the two upper thirds of the iliac fossa, by loose cellular tissue which is rarely continuous with fibrous tissue. From this disposition of parts, it results, that the pus surrounding the kidney will find its way equally easily into the cellular tissue which forms the immediate covering of the psoas muscle, or into the sub-peritoneal or sub-aponeurotic cellular tissue. This continuity of the cellular tissue of the perinephric region with that of the iliac fossa is the anatomical explanation of the facility with which perinephric abscesses, following the iliac and crural vessels, open into the triangle of Scarpa, or at the trochanter minor, following the psoas muscle to its lower insertion.

The perinephric adipose tissue is continuous with the cellular tissue of the lumbar region beyond the quadratus lumborum, between the margins of the latissimus dorsi and obliqus externus abdominis; that is to say, in the situation where J. L. Petit and Jules Cloquet have observed lumbar herniæ, and where it has been recommended to make the incision in nephrotomy. This continuity of the cellular tissue shows us the course taken by the pus in cases of perinephric abscess, when it is poured out into the subcutaneous cellular tissue of the lumbar region, to be there either localised, or spread out in the dorsal and gluteal regions. The lower surface and the substance of the quadratus lumborum are traversed by the lumbar arteries and veins, vessels of sufficient size to be sources of mortal hemorrhages when several of them are divided by the surgeon's bistoury.

The relation of the perinephric cellular tissue to the iliac fossæ, true pelvis, colon, diaphragm, and psoas muscle, enable you to understand the peregrination of these abscesses.

I now resume their consideration:

Before Rayer[1] wrote, little attention had been paid to perinephric abscesses. His researches were followed by those of Parmentier,[2] Féron,[3] Lemoine[4] and Ch. Hallé,[5] who collected a large number of cases from the lectures and practice of their teachers, among whom I would specially name Demarquay, Vigla, Gueneau de Mussy, and Chassaignac.

Perinephric abscesses arise from various causes, some of which are of a complex nature. Wounds in the renal region may occasion the formation of abscesses around the kidneys: Baudens mentions a case of this kind. Contusions in the lumbar region are still more common causes of perinephric abscesses. M. Bergounhioux (of Clermont) and M. Bienfait (of Rheims) each report a case showing the effect of a direct contusion in producing these abscesses.

In M. Bergounhioux's case, the patient was a peasant, who in falling from a tree, received a severe contusion in the right lumbar region. Extensive ecchymoses in that region, and hæmaturia lasting for some days, were the immediate results. The patient soon experienced deep-seated pain; and fever set in. The hæmaturia ceased; but the bruised region became swollen, there were frequently recurrent rigors, and ere long, fluctuation could be distinctly recognised. An excision made external to the sacro-lumbar mass, gave exit to a very considerable quantity of phlegmonous pus. In a few weeks, the patient, being quite cured, left the Hôpital Clermont-Ferrand.

In M. Bienfait's case, the patient was a nurse who fell down eight steps of the stair striking the edge of a pail. She was severely contused; but it was not till two days after the accident, that she took to bed with fever and vomiting. When M. Bienfait saw the patient, he observed that the decubitus was dorsal: the face was pale, anxious, and sickly looking: the pulse was small and quick. The hypochondrium and the right side were swollen, tense and painful. The urine passed since the previous evening was observed to have deposited a small quantity of blood. For three weeks, there was a continuance of fever, excitement, and night-delirium: there was also diarrhœa. At that time, the right side of the abdomen was much increased in size: there was great tumefaction of the lumbar region, and complete obliteration of the costo-iliac hollow; these parts were œdematous. The intra-abdominal tumor was so large as to be compared by M. Bienfait to the uterus at the sixth month of gestation: it was situated in the side, stretching into the hypochondrium. It had every indication of having an alcoholic character at the inferior surface of the liver, which organ it pushed upwards and forwards: it extended to the left beyond the umbilicus, and downwards, it reached to the upper part of the iliac fossa; and it transmitted to the flat hand, when placed over the loins, the impulse communicated to its anterior portion. Very obscure fluctuation was detected. Caustic potash was applied over the opening in the aponeurosis through which lumbar hernia takes place. Three weeks elapsed without there being any discharge of pus, the constitutional symptoms continuing as before. M. Bienfait made a puncture through the slough, when only a very small quantity of pus flowed out; but in a forty-eight hours afterwards, the discharge became very profuse, and the tumor collapsed. The case terminated favorably. I may mention that in this case, paralysis of the right leg existed for four or five days when the tumor was at its maximum.

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[1] Rayer:—Traité des Maladies des Reins. Paris, 1839.

[2] Parmentier:—*Union Médicale*, Vol. XV, année, 1862.

[3] Féron:—Thèse sur la Perinéphrite Primitive. Paris; 1860.

[4] Lemoine:—*Union Médicale*, 20 Juin, 1863: T. XVIII, p. 551.

[5] Hallé:—Des Phlegmons Perinéphrétiques, Thèse soutenue le 13 Août, 1863.

There are other cases in which no direct contusion, no blow on the loins has been received—cases in which violent exercise, such as a long journey on horseback, or the jolting of a badly hung carriage, have been sufficient to cause the formation of abscesses round the kidneys. This etiology can hardly be explained, except by admitting that the shocks imparted to the kidney by the trot of the horse, or the jolting of the carriage, have irritated the perinephric cellular tissue; but Dr. Hallé, while he admits that these causes in part determine the formation of the abscesses, correctly remarks, that at the same time the patients have been exposed to cold when in a state of perspiration. The majority of authors concur in recognising a chill as a cause which may at any moment act as the determining cause of perinephric abscess.

Violent strains seem sometimes to occasion perinephric abscesses. For the history of a case of this kind, we are indebted to Professor Tardieu and his *interne*, Dr. Aug. Ollivier. A workman employed in the plaster quarries, when lifting a heavy load, felt acute pain in the left lumbar region. The pain having diminished, he continued to work; but twelve days later, was obliged to take to his bed, and seek admission in the Hôpital Lariboisière. At that period, there was an even tumefaction of the lumbar region, unaccompanied by redness of the skin: the tumefaction was greatest on the left side: the swollen parts seemed œdematous. There were severe lancinating spontaneous pains shooting through the chest and abdomen. These pains made the respiratory movements painful, and occasioned very acute colics. Slight pressure made posteriorly hardly increased the pain, whilst it was aggravated by hard pressure. Fluctuation, though carefully sought for, could not be detected. There was no albumen in the urine. The skin was burning, the pulse was 110, and the thirst very great: there was loss of appetite, constipation, and no vomiting. Six days after admission to the hospital, this patient had fluctuation in the left lumbar region: from a deep incision made into the abscess, there flowed about a tumbler of greenish, creamy pus, in which were seen some sanguinolent striæ, but no muscular debris. The opening of the abscess gave almost immediate relief from pain. By introducing a probe into the wound, the exact seat of the abscess was ascertained to be behind the left kidney, neither extending above nor below the organ. It was necessary, some days later, to enlarge the incision, on account of a dread of purulent absorption from there not being a very free exit for the pus: gradually, the source of the discharge dried up; and within six weeks after admission to the hospital, the man had completely recovered. Gentlemen, it is important to remark, that in this case of primary perinephritis, the strain was enough—without any lesion of the kidney—to have produced the local inflammation: the patient was a man of good constitution, who never had had any serious illness, nor any urinary affection.

I have the details of another case similar to that now described, in which the influence of a strain in producing perinephric abscess is clearly established. The son of one of our most celebrated painters, a youth of twenty, felt acute pain in the loins, when straining to haul up a boat upon the bank of a river. The pain soon abated: but in some days, became so acute, that the young man had to take to his bed. The physicians and surgeons consulted were all of opinion that there existed perinephritis, which would be likely to terminate in suppuration.

I am indebted to the ordinary medical attendant, Dr. Bonin (of Poissy), for important information regarding the termination of this case; and as it supports a doctrine in general pathology to which I have often called the attention of my auditors, I ought not to neglect to lay it before you. I was informed that the perinephritis did not, in this case, terminate in suppuration; and that diminution of the pain, the predominating morbid phenomenon, sufficed to dispel all the other symptoms. The acute character of the pain caused the patient to utter piercing cries: in these circumstances, some drops of the solution of the neutral sulphate of atropine, injected into the cellular tissue of the lumbar region promptly relieved the pain, whereupon the patient was cured as if by enchantment. Does that mean, Gentlemen, that the diagnosis was a mistake, and that perinephritis never existed? That is not my opinion. The surgeons who had been called in, being experienced in such affections, could not have been mistaken: the beginning, the progress, and the cause of the affection presented characters too distinctive to admit of any doubt on the subject. The pain having been put an end to, all the other symptoms yielded, and the progress of the inflammation was arrested. You know, Gentlemen, the part which pain has in the inflammatory fluxion: how often have I demonstrated this to you at the bedside of the patient, particularly in cases of sub-orbital neuralgia! Have you not seen that when the pain ceased, all the other morbid phenomena disappeared in a few hours? It is very probable, then, that in the young patient whose case I have just now narrated, the cessation of all the symptoms of perinephritis is attributable to the cessation of the pain. You will, I think, be the more prepared to accept this interpretation of the course of the disease from my having previously pointed out to you that there is sometimes a spontaneous arrest, transient or permanent, of the symptoms of perinephritis.

It appears, then, that a strain may cause perinephritis: the two cases which I have just described, will enable you under similar circumstances, to foresee, from the commencement, the symptoms to be dreaded. When your attention is once directed to the existence of a deep-seated abscess, you ought carefully to seek, day by day, for fluctuation, and all the signs, local and general, of the formation of pus. You will thus be able to seize the opportune moment for opening the abscess, and preventing the pus from working its way into the iliac fossæ, and setting up very formidable symptoms.

I am now going to lay before you, Gentlemen, several cases in which the formation of the abscess was consequent upon a very slight cause; but that is not so always, and sometimes the existence of perinephric abscesses are not discovered till long after their beginning. MM. Cusco and Chassaignac have seen abscesses supervene several months, and even several years, after the action of the probable cause. In the cases to which I refer, the patients had received serious contusions in the lumbar region: the pain had disappeared: and it was not till a much later date, that, under the influence of a chill, or without any appreciable determining cause, the abscess showed itself. I am inclined to think—no new contusion having been received—that fatigue, a strain, or a chill aroused irritation, till then latent. In such cases, there are two periods in the formation of the abscess. There takes place, in the first period, as the result of the contusion, a slow, latent modification of the perinephric cellulo-adipose tissue; and in the second period, we have the determining cause in operation—the fatigue, strain, or chill—when pus is formed, and the local and general symptoms attendant on suppuration show themselves.

Perinephric abscesses very often have as their starting point nephritis or calculous pyelo-nephritis. In such cases, the inflammation may extend, by contiguity, from the kidney to the surrounding cellulo-adipose tissue: at other times, calculi impacted in the calices, the pelvis of the kidney, or the ureters, produce inflammation and ulceration of the different parts of the urinary apparatus, and when fistula is formed, they give rise to urinary abscesses which ought to be opened with the least possible delay. In these cases, the abscess is generally preceded by nephritic colics, and disturbance of the urinary function: it is not unusual to discover, on exploration with a probe, the presence of calculi in the abscess itself: at other times, the calculi remain imprisoned in the kidney, and it is not till after some time has elapsed, that the calculi present themselves at a fistulous opening of the abscess. Some have recommended search for the calculi. Dr. Miguel has even applied lithotrity to imprisoned calculi of too large a size to traverse easily the urinary fistula. At the present date, however, surgery takes a less active part in the extraction of renal calculi: the present practice is to wait till they present themselves at the mouth of the abscess.

Perinephritis is also, you see, a symptomatic affection, due to the presence of foreign bodies tending towards elimination: in some rare cases, the foreign bodies are hydatids situated in the perinephric cellulo-adipose tissue, in which they cause suppuration. Cases of this kind have been observed by Dr. Rayer, and Professor Denonvilliers, in patients in whom (as clinical examination had not given any reason to suspect hydatids in the liver, lungs, pleuræ, or other parts of the body) it was impossible to diagnose their presence in the perinephric region.

As the purulent diathesis may affect any part of the organism, it is very natural to suppose, that it may cause abscesses to form in the cellulo-adipose capsule of the kidney. In the *Edinburgh Medical and Surgical Journal* there is reported a case of perinephric abscess in a sailor, suffering from the affection popularly known at Plymouth as the "disease of the docks." This disease, according to Butler, is a fever, which may lead to purulent formations in different parts of the cellular tissue. Dr. Duplay reports a case of perinephric abscess, consecutive to an attack of typhoid fever, treated in Dr. Pelletan's wards in the Hôpital de la Charité. Dr. Desruelles and Destouches discovered a perinephric abscess in a woman of sixty, convalescent from gangrenous pneumonia.

In these cases, there is probably a special alteration of the humors leading to the formation of numerous abscesses, as is so frequently observed after small-pox.

Again, the *puerperal state* predisposes to the formation of pus, even when no puerperal epidemic is prevailing. As you know, mammary and iliac abscesses are of very frequent occurrence during the first months after delivery. You also recollect, how often I have called your attention to the pain which recently delivered women experience in the lumbar regions: this pain, and the pain in the uterine appendages, is not felt by patients unless pressure be made over the loins: you saw two cases of this kind in Saint-Bernard's ward; and in them, after some days, I discovered perinephric abscess. Bear in mind, that these abscesses around the kidney are not always the consequences of propagation of an abscess situated in the broad ligament or the iliac fossæ. The first of the two patients to whom I have now referred, who occupied bed 25 bis, Saint-Bernard's ward, had a chill on the fourth day after delivery, the symptoms being rigors, fever, and pains in the abdomen. All these symptoms subsided under the influence of rest: fifteen days later, this woman, having committed imprudences, was seized with shivering and fever, and a renewal of abdominal pain in the sub-umbilical region. The pain extended to the left iliac fossa; and some days later, by digital examination, I was able to detect an abscess of the broad ligament, which might open any day into the vagina, and so completely disappear. But when my attention was occupied in following the progress of the abscess in the broad ligament, the patient was seized with pain in the right side: pressure in the lumbar region increased the pain: in the renal region, there existed a decided doughy fulness, most perceived when the region was grasped between the two hands. The liver was not painful on percussion, and did not extend beyond the false ribs: the right iliac region was not swollen, nor was it painful on palpation. It appeared, then, that the lesion was limited to the right renal region; but, gradually, the pain and fulness diminished, the fever moderated, and there was no recurrence of the rigors: ere long, by palpation and percussion, I ascertained, that the inflammation was undergoing resolution. Two months after admission to the hospital, this patient left it perfectly restored in health.

This termination of perinephric abscess by resolution is unusual; and the case of the patient, bed 4 Saint-Bernard's ward, is one proof more that suppuration is the rule. Though this woman had a natural labor, the uterus remained painful and very bulky: the uterine appendages were much inflamed, and the pus formed in the right broad ligament had two outlets, one into the bladder, and the other into the vagina. For fifteen days after the spontaneous opening of the abscess, everything went on to a wish; but at the end of that period, the patient was seized with rigors, fever, and pain in the right side. The liver was bulky, and descended within three finger breadths of the crest of the ilium. The mobility of the liver was ascertained by making the patient respire. In the lateral parts of the abdomen, and in the loins, there was felt an engorgement which could only be referred to the renal region. The swelling occupied the whole of the costo-iliac hollow. By placing one hand behind, and another in front of the tumor, the movements of the liver during inspiration and expiration could be felt: and at the same time, a tumor was perceived which did not move during inspiration. There was no reason to suppose that this tumor was stercoral: no sign of occlusion of the intestine existed: the iliac fossa was free, and was not the seat of pain. The initiatory rigors, fever, and lumbar pain suggested the probable existence of a perinephric abscess. As the fever was still going on, it seemed best to wait:

by-and-by, detecting fluctuation, I opened the abscess external to the sacro-lumbar muscular mass: as soon as I reached the deep layer of muscles, I laid aside the bistoury for a hollow sound, with which I tore through the tissues: forthwith, there came a gush of non-fœtid pus from the aperture I had made. On introducing the finger into the abscess, I could feel the kidney. Poultices were applied to the loins, whereupon the flow of pus was easy and abundant. With the view of drying up the abscess, and modifying the action of the suppurating surfaces, I several times employed injections containing iodine: little by little, the flow of pus diminished, and the incision began to cicatrise, when the patient was again seized with rigors, and pains in the right iliac fossa, thigh, and knee. Soon afterwards, an abscess was detected in the iliac fossa. Finding that this abscess had a tendency to pass below the crural arch, I begged Professor Jobert (of Lamballe) to open it. He made an incision one finger's breadth above the arch, and in a line parallel to it. A great quantity of greenish pus issued from the opening: the cavity of the abscess was washed out with water containing tincture of iodine: the pain of the psoïtis continued for a long time, the inferior extremity, nevertheless, resuming its normal position. There was reason to hope for a permanent amelioration, when the patient was seized with diarrhœa, and then with hectic fever: but she died some weeks after the opening of the iliac abscess. It was impossible for me to obtain a *post-mortem* examination of the body. This is much to be regretted; for we could then have ascertained whether the perinephric abscess (as is exceedingly probable) had worked its way to the iliac fossa. You can understand how the pus collected round the kidney, by following the psoas muscle, might reach the cellular tissue of the iliac fossa below the aponeurosis.

At other times, the pus may filter through the layers of aponeurosis, invading the cellular tissue which lines the peritoneum of the renal and iliac regions: in these cases, the abscess is not in immediate contact with the psoas muscle. Anatomy fully explains the infra-aponeurotic and supra-aponeurotic position of abscesses in this region. It must, however, be remarked, that the numerous aponeurotic openings allow the pus situated below the iliac aponeurosis to invade the sub-peritoneal cellular tissue.

Before leaving this part of my subject, I must remark, that in those cases in which perinephric abscesses have been complicated with pleurisy or pleuro-pneumonia, the thoracic affection and the perinephric abscess have always been on the same side. It was so in the cases observed by Desruelles, Cazalis, Demarquay, and Bernutz: in these cases, the pleurisy and pleuro-pneumonia evidently depended on vicinity.

There are other causes, besides those I have mentioned, which lead to the formation of abscesses around the kidney. Pain, as you are aware, which is most commonly the consequence of inflammation, may likewise be the cause of an inflammatory fluxion. Paludal sub-orbital neuralgia is often accompanied by hyperæmia of the conjunctiva and a profuse secretion of tears. If the orbital congestion continue for some time, a copious secretion of mucus is induced from the palpebral glands; and should the congestion last still longer, a certain amount of muco-purulent secretion will be seen at the angle of the affected eye. The pain may, under such circumstances, although there exist no primary local lesion, occasion inflammation, which will disappear as soon as the paludal poisoning of the system has been modified by general specific treatment. It is very evident then, that in such cases, pain may induce inflammation of the eye. Toothache, also, often determines a fluxion to neighbouring tissues. Some neuralgic affections of the cervix uteri, not attributable to any organic lesion of the uterus, determine, during each paroxysm of pain, a secretion from the follicles of the cervix, and chronic inflammation of the mucous membrane of the cervix. We have seen how paludal poisoning, a general cause, may induce inflammation, and have also seen how neuralgia originating in a local cause, may lead to a similar result. You will find other cases recorded in which pain has induced inflammation at a distance from the seat of pain. Dupuytren mentioned in his clinical lectures, that having included one of the branches of the brachial plexus in ligature of the axillary artery, he found, on making the *post-mortem* examination of the patient, that there was an abscess of the brain; and he was inclined, in that particular case, to think that the constant acute pain endured by the patient during the continuance of arterial ligature, was the determining cause of the cerebral abscess. To me, however, this explanation seems rather hypothetical.

Pain may similarly explain the formation of some perinephric abscesses. After attacks of severe nephritic colic, it is not unusual to meet with perinephric abscesses, the opening of which shows that the formation of pus could not have been the consequence of a urinary fistula; while the examination of the urine has proved the non-existence of purulent nephritis. In these cases, therefore, there is no ground for supposing that inflammation had been propagated from the kidney to the surrounding cellular tissue, and we certainly must in these cases attribute to pain a large share in the formation of these abscesses. Again acute pain of the bladder may induce the formation of abscesses around the kidney. At the close of the year 1862, I was called in, in consultation, by Dr. MacCarthy, my very honorable colleague, to visit a lady who had complained for more than two years of a very inconvenient irritability of the bladder. An irresistible desire to urinate was excited by the presence of even a few drops of urine in the bladder. On October 4th, 1862, this lady wore, for a few hours, a very tight dress. On the following day, she took to her bed, and complained of acute pain in her right side: the pain extended into the lumbar region. She had had no stool for three days. She had no fever. On October 8th, the menses appeared, and continued for twenty-four hours. The pain in the right side continued. For eight days, the local pain went on increasing. She had rigors and fever, increasing in severity day by day. No relief was obtained from poulticing, and two applications of leeches. Every day, care was taken that the bowels should be opened by the use of an injection, or aperient pills; but notwithstanding, there was no abatement of the pain and fever. On October 15th, for the first time, my honorable colleague when making pressure on the seat of pain, detected a hard round tumor. The pulse was rapid, the skin hot; and the shivering constantly recurrent. It was at this period, that Dr. MacCarthy consulted me in the case. We were both satisfied that there was a lumbar tumor: by palpation, I ascertained that the movements of the liver during respiration were independent of the tumor, which remained immovable. The tumor was large, and very painful. Dr. MacCarthy and I took exactly the same view of the case; and were of opinion, that the patient had a perinephric abscess. Although the patient had had hepatitis in Bombay, there was no ground for supposing that she had a tumor of the liver. That she had perityphlitis, was an equally untenable hypothesis, because that is an inflammatory affection generally seated in the iliac fossa: moreover, there was no pain in the large intestine, and the stools presented no important alteration. In consideration of the fever, and the size of the abscess, our prognosis was given under reserve; but we held that the patient had perinephritis, independent of any lesion of the kidney, and depending, probably, on sympathy with a constantly irritable bladder. The immediate cause of the affection might have been a chill, or excessive compression of the lumbar region by a too tight corset or gown.

The tumor rapidly increased in size, the rigors recurred, there was complete loss of appetite, and a twofold severity of the fever. Under these circumstances, Dr. MacCarthy, considering the time had arrived for opening the abscess, again called me in to consult on the case, and, along with me, summoned my honorable hospital colleague, M. Alphonse Guérin. When we met, the tumor occupied the whole of the right side, extending to the umbilicus. The fluctuation was very obscure; and the slightest pressure occasioned extreme pain. There was well marked œdema in the right lumbar region. Hesitation as to the course to pursue was impossible: it was evidently necessary, without delay, to afford an outlet to the pus, so that the abscess might not have time to open into the intestine, or work its way into the iliac fossa. M. Alphonse Guérin made an incision ten centimeters in length near the external margin of the sacro-lumbalis muscle. Having reached a depth of four centimeters, he laid aside the bistoury, and with the index finger of the right hand penetrated the abscess, whence issued a torrent of fœtid thick pus. For four following days, the patient had a continuance of slight fever with shivering. So that there might be no doubt as to the pus, which was still somewhat fœtid, having a free outlet, the opening into the abscess was enlarged by the finger. The discharge gradually decreased, and lost its odour. Eight days after the opening of the abscess, there was a notable amendment in all the symptoms: the suppuration was inconsiderable, the wound was cicatrising, and the appetite was restored. No check occurred in the progress towards recovery. On the eighteenth day after the operation, the wound was perfectly closed. At that date, there was no trace of the tumor; and some days later, the cure may be said to have been complete.

Is it necessary, Gentlemen, that I should expatiate upon the leading symptoms in this case? Habitual irritability of bladder, persistent and increasing pain in the lumbar region, then fever, soon afterwards, pain in the renal region; and finally, a tumor and abscess were the phenomena.

In conjunction with this case, let me call your attention to another, also bearing testimony to the influence of pain and irritability of the bladder in producing perinephric abscesses. In the latter months of 1863, a great personage was operated on for vesical calculus by Civiale. Lithotrity was successfully performed; and the patient was able to leave Paris to recruit his strength in the country. But some days after the operation, the illustrious patient began to feel pain in the renal region, in one side only. Fever, rigors, and loss of appetite supervened. Several surgeons were assembled in consultation: after ascertaining that there was no symptom of lesion of the urethra, bladder, or kidney, they were disposed to attribute the lumbar pain to ilio-lumbar neuralgia consequent upon the lithotriptic manipulations. The pain continued for several weeks in the renal region, and extended into the iliac fossa of the same side; but in the latter situation, there was neither tumor nor any sign of psöitis. The fever and rigors, nevertheless, continued; and the patient went on losing strength. M. Nélaton was now asked to join the other surgeons in consultation. Informed of all the symptoms which I have now related to you, and of the conditions under which they showed themselves, the learned professor very attentively explored the renal region. Palpation occasioned pain; and there was a slight fulness in the lumbar region, but (as the matter was still very deep-seated) fluctuation could not be perceived. M. Nélaton had no hesitation in stating that there was a perinephric abscess. A large incision was made at the margin of the quadratus lumborum muscle, and immediately there was a gush of healthy pus without special odour, and not containing any clots of blood. By introducing the finger deep into the wound, it was ascertained that the abscess waa situated in the circum-renal cellulo-adipose tissue. From the day of the operation, both the fever and pain disappeared. There was no recurrence of the rigors: day by day, the appetite returned: and at the present date, recovery may be said to be complete, although for some weeks, the patient had had a rather obstinate diarrhœa. There was neither renal nor vesical lesion. It is, therefore, very probable, that the abscess was caused by the irritation of the bladder, acting by sympathy upon the perinephric cellulo-adipose tissue.

Let me now quote, from Chopart's treatise, a case which I consider very interesting, inasmuch as it is one more fact to prove the part which pain has in producing perinephric abscess:—

"I saw a man," says Chopart, "who had had his right testicle amputated for cancer: All went on favorably till the thirty-second day after the operation, when he had considerable rigors, and complained (for the first time) of heat and lancinating pain in the kidneys. The wound—the cicatrisation of which was complete—became pale and dry. The fever continued. Next day, the abdomen was tense. During the night, the patient had nausea, and restless excitement. On the following day, he died. I was present at the *post-mortem* examination. There was an abscess in the adipose tissue of the left kidney; the pus was serous and fœtid: the cellular tissue of the spermatic vessels was infiltrated by the same matter: and there were also two small abscesses in the pelvis, on the same side. As the whole of the spermatic cord had been included in the ligature, in place of only the spermatic artery, it was supposed that the ligature might have occasioned the suppuration by the irritation it had excited in the cellular tissue of the pelvis and loins of that side; and of which irritation the patient had given signs at the moment the ligature was tightened, for he then complained of an acute pain in the region of the left kidney which continued for several hours. All other parts of the body were in a healthy state."

It would certainly be difficult to find in the annals of science a case in which the influence of pain was more distinctly marked. There was not the slightest grounds for supposing that there was either phlebitis or purulent infection; for, on the one hand, the pus found in the course of the cord, was not in the veins, and on the other hand, the patient never had had a metastatic abscess. It appears to me that in this case, pain caused irritation of the cord and was re-echoed, if I may so speak, by the cellular tissue of the kidney, just as gonorrhœal irritation of the urethra may make itself known in the joints, and produce blenorrhagic arthritis. It is, therefore, most important to remember, that there is a special irritation in blenorrhagia which may cause blenorrhagic arthritis; and also, that irritation of the bladder or spermatic cord may act sympathetically upon the perinephric cellulo-adipose tissue.

I have now to tell you that there are cases in which the perinephritis cannot be attributed to any of the causes we have been considering. In the patient who occupied bed 2, Saint-Bernard's ward the cause of the perinephritis could not be ascertained. The same impossibility to discover the cause of the affection existed in a case to which I was called in consultation with Dr. Cavasse in October, 1861; and of which the following is a summary of the details:—M. X., a man aged thirty-five, since an attack of typhoid fever, had been in rather feebler general health than previously, but was able, nevertheless, actively to attend to his business. On his return one day from hunting, when he had fatigued himself by overwalking, he complained, for the first time, of pains in the left lumbar region. The affection, supposed to be lumbago, was attributed to damp weather. Every time the patient bent forwards, pain was felt. He had had neither fever nor rigors; and his appetite was unimpaired. He continued to go about his ordinary occupations. The pain, however, was constant. Eight days after his first visit, Dr. Cavasse made a renewed examination of the seat of pain, when, not without surprise, he found a tumor in the left lumbar region. In that situation, there was slight redness of the skin, and doughiness of the cellular tissue. On endeavouring to make out the limits of the tumor by the finger, a hardness of from seven to eight centimeters was discovered. The tumor was as large as a hen's egg, and was prominent under the skin. It was five or six centimeters from the vertebral column. Fifteen days later, the tumor became more prominent: both the redness and doughiness increased. The patient had lancinating pains; and, by palpation, deep, obscure fluctuation was detected. Observe, Gentlemen, that up to this date, there was neither fever nor loss of appetite. These were the circumstances in which Dr. Cavasse asked my advice. I made out all the signs of a deep-seated abscess in the lumbar region. The patient's age, his usual state of health, the absence of any osseous lesion in the ribs, vertebral column, or pelvis, excluded the idea of there being any indolent abscess, the result of congestion. The pain in the region, the redness of the skin, and the fulness of the cellular tissue indicated the existence of acute inflammation: the deep seat of the abscess was the cause of the tardiness with which fluctuation became appreciable. What were the original seat, and cause of the inflammation? There had been neither intra-abdominal pain, nephritic colic, nor any noteworthy alteration in the urine. The kidney, consequently, could not be the cause of the condition I have now described: the patient had not received a blow in that region. It was therefore very difficult to determine the cause of the abscess. But the pain at the first, its persistence in the renal region, and the swelling and heat in that situation, were sufficient indications that the evil had originated there. Though the kidney was not in any way the cause of the affection, and though no direct traumatic cause was in operation, there was every reason to believe that the perinephric cellulo-adipose tissue was the seat of the suppuration, and that there existed a primary perinephric abscess. That was my opinion when I first saw the patient with my honorable colleague, Dr. Cavasse. I however recommended my colleague to delay opening the abscess till the matter became more superficial. On an early day of November, that is to say, five or six weeks from the beginning of the affection, M. Cavasse opened the abscess: there flowed from the incision a tumbler of non-fœtid pus mixed with blood. The walls of the abscess were hard; and its cavity presented numerous anfractuosities. The opening was kept patent by the introduction of a dossil of charpie, so that there was an easy flow of pus. Injections containing tincture of iodine were employed with a view to modify the surface of the interior of the abscess. The cure was not completed till the middle of January 1862.

This case is interesting for more reasons than one. First of all, it is interesting, because it proves that a primary perinephric abscess may exist without any other appreciable cause than excessive walking, or damp weather: and in the second place, it shows, that a deep seated perinephric abscess may be slowly developed without causing the constitutional symptoms which usually occur in similar circumstances.

Other causes, besides those which I have mentioned, give rise to perinephric abscesses.

In a previous lecture, I remarked, that pleurisy or hepatitis is a very frequent sequel of severe hepatic colic; and that cellular adhesions between the liver and diaphragm are very common in persons who have recently had severe hepatic colic. In the cases in which I pointed out to you that these colics were succeeded by pleurisy of the right side, I explained the phenomenon by telling you, that the peritoneal inflammation was communicated to the diaphragm, and from the diaphragm to the pleura. I also reminded you of a very usual anatomical disposition, a separation of the muscular fibres of the diaphragm, by which the peritoneum and diaphragmatic pleura are brought into contact, and are adherent, being separated only by a very thin layer of cellular tissue. It is easy to understand, that when such conditions exist, inflammation of the peritoneum may very easily be propagated to the pleura.

You can now understand how inflammation of the gall-bladder, so common in hepatic colic, may be propagated to the peritoneum covering the right kidney, and how that inflammation may be the starting point of a perinephric abscess.

Perforation of the bladder by a calculus, which usually produces rapidly mortal general peritonitis, may (as in a case I described to you, which we had an opportunity of observing in Saint-Bernard's ward) determine adhesions between the bladder and neighbouring parts; and then, the calculus escaping from its position, falls into adventitious cellular tissue, which will patiently support its presence till the abnormal contact excites inflammation of the perinephric cellular tissue. It was probably a case of this kind which I had an opportunity of observing in the practice of my colleague Dr. Millard. We saw together an old lady suffering from attacks of hepatic colic. After one of these attacks of unusual duration and severity, she had all the symptoms of acute hepatitis, accompanied by inflammation of the gall-bladder. There was very intense pain in the sub-hepatic region: there was fever and severe general disturbance of the system, when all at once the pain extended to the right renal region: a large tumor then made its appearance, accompanied by rigors, and soon afterwards, by undoubted signs of perinephric abscess. The contents were evacuated by an opening and counter-opening made by Dr. Trélat. The symptoms rapidly subsided after the operation.

We believed, that inflammation of the gall-bladder was the cause of adhesions forming between the cholo-cystic peritoneum, and the peritoneum which covers the intestines and extends above the kidney: that a calculus had escaped into the adventitious cellular tissue: and that the inflammation had been propagated to the perinephric tissue. This, of course, was only an hypothesis: but the statement is not hypothetical, that the hepatic colic and consecutive inflammation of the gall-bladder and peritoneum were the cause of an abscess forming in the circum-renal cellular tissue.

The symptoms of perinephric abscesses vary with the cause—according as they follow disease of the kidney, or supervene under the influence of some other cause. In the first instance, the symptoms of perinephritis are preceded by the peculiar pain of nephritic colic: there may have been calculous nephritis; and sometimes gravel and calculi are found in the urine. Hæmaturia may have existed in some cases; and should inflammation have invaded the calices and pelvis of the kidney, more or less pus will be deposited by the urine. Should pain, swelling, redness, and doughiness supervene in the lumbar region, it will be natural to ascribe the perinephritis to lesion of the kidney.

Generally, however, the perinephritis occurs irrespective of any renal lesion. Generally, quite suddenly, and as the result of very various causes, the patient complains of a deep-seated, diffuse pain, which may be acute or dull, in the lumbar region. This spontaneously originating pain, which is sometimes shooting, is always increased on pressure, particularly when the seat of pain is pressed between the two hands. The pain is sometimes absent for weeks or months, not returning till a new determining cause arises. Generally, however, the pain is persistent, and goes on increasing till the pus is evacuated. This pain is always a very important symptom, because for several days, or weeks, no other local symptom may exist. The general disturbance of the system, however, shows that the pain depends on an organic cause: the patients have continued fever, with fits of shivering in the evening. Every day, the patients have rigors, followed by hot and sweating stages. They soon lose appetite, and rapidly become thin. Sometimes, the febrile paroxysm sets in with vomiting; and it is almost always attended by obstinate constipation.

Within a period, then, varying from eight to fifteen days, the patients present no symptoms except local pain, general debility, and quotidian ague. Afterwards, other local signs of deep-seated inflammation show themselves: the pain becomes more and more decided on making pressure over the region, in which also there is more or less doughiness: the costo-iliac hollow is effaced; and if (the patient lying on his back) the physician press his hand deeply into the lumbar region, he perceives by the touch, as well as by the eye, that a more or less marked projection exists; and if, at the same time, he place the other hand upon the corresponding anterior region, he recognises, between his hands, a deep-seated tumor continuous with the subcutaneous cellular tissue. This tumor remains fixed during full inspiration and expiration, performed at the request of the physician—a fact which establishes with certainty, that the tumor is independent of the liver, which rises and falls with each inspiratory and expiratory movement. The doughiness of the lumbar region is often accompanied by œdema, and this œdema may extend to the dorsal region and hips. There is sometimes also, a little redness of the skin. This redness is erysipelatous in cases in which the abscess extends into the cellular tissue of the region. From the very commencement of the local signs of inflammation, fluctuation can be distinctly felt. As, however, it is very deep-seated, it requires great aptitude on the part of the examiner to detect it with certainty: its existence may sometimes only be suspected from the complications of œdema, doughiness of the region, and from the general symptoms. As soon as the formation of pus commences, there is an exacerbation of the fever: the pulse acquires a certain degree of fulness, and becomes harder and more resistent, while, at the same time, the patient complains of frequent shiverings. Under these circumstances, the indication to evacuate the pus is quite clear: there must be no hesitation in opening the abscess, for if this be delayed, the pus may work its way into the iliac fossa and coxo-femoral articulation, thereby endangering the life of the patient.

Though, sometimes, the result of perinephric inflammation is the formation of a partially encysted abscess—the abscess remaining limited to the perinephric layer of fat, and showing no disposition to extension beyond the lumbar region—yet, at other times, the inflammation gains the cellular tissue of the neighbouring parts, invading perhaps the sub-diaphragmatic cellular tissue, occasionally even passing that barrier and reaching the pleura or lung, ending there in pleurisy or pneumonia. These are the terminations and complications which result from too long delaying surgical intervention. This has been observed by MM. Demarquay, Cusco, Cazalis, and Bernutz. The pus sometimes penetrates even into the bronchial tubes. A case is reported by Dr. Rayer, in which there was a vomica in the lung attributable to no other cause than a perinephric abscess.

The inflammation often invades the iliac fossa, the patients then complaining of pain in that region; and if an outlet be not afforded to the pus, a tumor is soon perceived projecting above Poupart's ligament, and passing below that ligament to show itself at the base of the triangle of Scarpa. In the latter case, the pus has followed the sheaths of the iliac and femoral vessels: at other times, it takes the course of the psoas muscle to the trochanter minor, and may, as we have seen, invade the coxo-femoral articulation.

I have already told you, Gentlemen, that the pelvic cellular tissue may be invaded by inflammation which had the renal region as its starting point. In one of our patients in Saint-Agnes's ward you saw that the pus had worked its way from the renal region to the cavity of the pelvis, and had then been evacuated into the bladder and vagina. The same patient had had double perinephric abscess: the abscess on the right side had terminated in resolution. In cases in which the pus has worked its way from a distance, it causes great structural damage, and gives rise to very long protracted suppurations, so that death is almost always the result of these migratory abscesses. The right practice, therefore, is to open perinephric abscesses as soon as the local and general signs of their existence are undoubted. You have seen, Gentlemen, that the spontaneous evacuation of the pus by the vagina or bladder may be a favorable termination of the case; but that mode of evacuation does not always occur: when the spontaneous evacuation takes place by the colon, the result may be unfavorable, as was seen at the autopsy of a patient who was treated in Professor Cruveilhier's wards, the details of whose case have been reported by Dr. Parmentier.

I know of only one case in which there was probably a spontaneous opening of the perinephric abscess into the peritoneum: perhaps some other cases may have been recorded. The rarity of such cases will be at once explained, when you consider the relations of the adipose envelope of the kidney to neighbouring organs and to the peritoneum. Perinephric abscess is generally situated behind the kidney: it is then separated from the peritoneum by the kidney; and the colon, in contact with the anterior surface of the kidney, also increases the distance between the inflamed cellular tissue and the peritoneum. Should the inflammation have a tendency to extend to the peritoneum, peritonitis will be induced, the result of which will be the formation of false membranes increasing the thickness of the peritoneum. The autopsies show, that under these circumstances the pus burrows under the serous membrane, and does not perforate it.

Allow me to make some further remarks upon the progress of perinephric abscesses, before I discuss the differential diagnosis between them and other affections. I have generally observed that the abscess has a tendency to make its way to the lumbar region, the inflammation gaining the different tissues little by little, till at last it reaches the subcutaneous cellular tissue. But should there be delay in opening the abscess, it will dissect the subcutaneous cellular tissue, and extend to the hips.

In 1861, bed 8, Saint-Agnes's ward, was occupied by a man forty-four years of age, who, when admitted to the hospital, had an abscess occupying the dorsal and lumbar regions of the left side. The man had fever, and an absolute loss of appetite: an erysipelatous redness covered the whole of the region invaded by the abscess.

The patient, who for six weeks had been suffering from lumbar pain, stated that he had been three months of the previous year under treatment for a similar affection in the Hôpital Saint-Antoine under the care of my lamented colleague the late Dr. Aran. In reply to repeated questioning, he affirmed that no incision had ever been made. He left the hospital without being cured, and for four months afterwards was unable to resume his work. He said that he did not know how the tumor had disappeared: he only knew that the improvement in his condition had occurred very gradually. He maintained that he never had had nephritic colic, and had never passed gravel or calculi with his urine. In the absence of positive facts as to his previous history, we had to rest satisfied by ascertaining the condition he was in when admitted to St. Agnes's ward. I have said that the pain had continued for six weeks: from the very first, there was fever; by degrees, an enormous phlegmonous tumor had formed, occupying the lumbar region, and subsequently extending to the dorsal region and hips. Palpation occasioned great pain; fluctuation could be detected in a salient point in the lumbar region. There was one point at which the tumor projected into the abdominal cavity, and extended in one direction from the liver to the iliac fossa, and in another to the umbilicus. The tumor was evidently a large collection of pus. By making an opening in the lumbar region, there was evacuated a large quantity of greenish yellow horribly fœtid pus, mingled with a fluid consisting of mixed pus and blood. About two litres of this fœtid pus were collected: and for several days after the operation, there was a great additional discharge of similar character seen on the poultices in which the region of the tumor was kept enveloped. Great relief followed the opening of the abscess: the fever abated: from the third day, the erysipelatous redness disappeared: the walls of the abscess progressively and rapidly contracted. In the external iliac region, there was another abscess which opened spontaneously: it discharged pus of the same character as the lumbo-abdominal abscess. By cautiously introducing a probe into the second abscess, it was ascertained that there was no morbid state of the iliac bone. By degrees, all the affected parts became cleansed, the fever disappeared, the appetite returned; and, to the great satisfaction of everybody, who had ascertained the extent of the affection, the patient was restored to perfect health in three weeks.

Lessons may be deduced from this narrative, notwithstanding its enormous gaps. What are they? We learn in the first place, that the abscess may extend exceedingly in the abdominal cavity without bursting the peritoneum, without reaching the iliac fossa, or without emptying itself into the large intestine. When by invading the tissues step by step, the abscess seeks to evacuate itself externally, it may detach these tissues, layer by layer, and so produce a subcutaneous or submuscular abscess, as happened in the case of our patient. That case also afforded an example of two perinephric abscesses occurring in the same person, and in the same side, at an interval of some months. This recurrence of the affection on the same side would indicate the continuance of some local cause, such as has been noted by observers. In our patient, however, we could not ascertain the existence of calculi in the kidney: moreover, the patient affirmed that he had never perceived any important change in the urine, and had never had nephritic colic.

It must be remembered, however, that calculi may remain a long time in the parenchyma of the kidney without occasioning acute pain. Pozzi, quoted by Rayer,[1] mentions the case of a man in whom the right kidney (as large as the head of a two years' old child, and weighing two pounds and a half), contained a calculus, the point of which had come through the renal parietes, occasioned gangrene, and led to the formation of a deep-seated abscess. The other kidney contained at least a hundred other calculi. "Sed quod mirum est," says Pozzi, "toto tempore vitæ nunquam conquestus est de doloribus nephreticis, calculis, urinis, sabulosis aut difficulter vel diminute fluentibus." Taking into consideration cases of this kind, we are justified in concluding, that our patient had renal calculi which gave rise to the perinephric abscesses.

I have sometimes seen a perinephric abscess occasion a very extensive lumbar phlegmon: it also, sometimes, happens that while these purulent collections detach the lumbar tissues from one another, emphysema of the entire dorsal region is produced. Twice I have seen this take place: the abscesses were opened; and exit was afforded to pus and fœtid gas. In one of the cases, the abscess communicated with the intestine: the patient passed pus with the stools, and from the incision there was a discharge of yellow matter, which certainly came from the intestine. The relation of the colon to the perinephric abscesses explains these occurrences.

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[1] Rayer:—Traité des Maladies des Reins: T. III, p. 35.

We now come, Gentlemen, to consider the important question of the diagnosis of perinephric abscesses. There are three morbid conditions which may serve as a basis of diagnosis: I refer to pain, tumefaction of the lumbar region, and fever. At the beginning of the formation of perinephric abscesses, the only symptoms are pain in the loins and fever. When the pain is on the right side, and when, associated with it, there is continued fever having a quotidian exacerbation, and prostration of strength, one may for a moment regard the case as one of typhoid fever: but the progress of the disease, and the absence of other symptoms peculiar to dothienteritis, soon rectify such a mistake.

Simple neuralgia does not generally cause fever: nor is fever induced by the pain of lumbago, which usually has its seat in the two sacro-lumbar muscular masses. On the contrary, the duration of the pain, its characteristics, and its being excited by pressure when there is perinephric inflammation, and also the continuance of the fever with its quotidian paroxysms, render it sometimes possible to diagnose the probable existence of perinephric abscesses, even in their first stage.

Nephritis and calculous pyelo-nephritis are generally preceded by nephritic colic: they may be accompanied by fever, and a saburral condition accompanied by vomiting and lumbar pains exasperated by pressure; but examination of the urine, which is frequently albuminous during the crises, and the immediate relief experienced on the calculus reaching the bladder, demonstrate, that the lesion is limited to the kidney and the excretory urinary organs. The diagnosis, however, will be more difficult in a case of pyelo-nephritis accompanied by a tumor in the lumbar region if repeated examination of the urine disclose neither the permanent nor the temporary presence of more or less pus in the urine. There are cases, however, in which there is still greater difficulty in diagnosing pyelo-nephritis accompanied by a tumor: I refer to those cases in which, on examination after death, a calculus has been found impacted in the urethra obstructing the passage of pus into the bladder. In those cases, the examination of the urine gives a negative result: but it is necessary to remark, that the distension of the pelvis and calices of the kidney may cause perinephric abscess, in consequence of the inflammation being propagated to the surrounding cellulo-adipose tissue, or from its forming a blind fistula leading to the effusion of pus and urine into the fatty covering of the kidney after which soon appear all the signs of genuine perinephric abscess.

Let me now describe to you, Gentlemen, a case of chronic perinephric abscess with pyelo-nephritis, which gives support to the remarks I have now made. For the history of this case, I am indebted to the courtesy of Dr. Demarquay.

The patient was a man, of about thirty years of age, previously subject to nephritic colic, who had passed small urinary calculi, and experienced pain in the right lumbar region for four or five years. When my honorable colleague saw him the first time, about the end of July 1864, he ascertained the presence in the right hypochondrium of an enormous tumor, having for its boundaries the liver, the iliac fossa, and the linea alba. This tumor was specially prominent on the anterior abdominal wall, the costo-iliac hollow was effaced, but the lumbar region presented neither deformity nor œdema. The liver did not appear to be implicated: no pain had ever been experienced in the hepatic region: nor had there ever been jaundice. There was fluctuation in the tumor. The urine deposited a considerable quantity of purulent mucus; and attacks of nephritic colic were followed by excretion of urinary calculi. From all these facts, the surgeon was led to believe, that the fluctuating tumor was an abscess in the circum-renal tissues. Dr. Demarquay opened the abscess by applying caustic potash to the most prominent point of the tumor. For several days, a great quantity of pus flowed from the opening: then, by degrees, the tumor diminished in size, its walls contracted, and there was reason to hope for a speedy cure, when the patient, after certain imprudences on the occasion of the fêtes of 15th August, succumbed under symptoms of very acute peritonitis. At the autopsy, the tumor was found to be really situated around the kidney: on the surface of the kidney, there were traces of chronic inflammation. The calices, pelvis of the kidney, and the urethra were full of pus; but it was impossible to discover any trace of urinary fistula: the pus could pass drop by drop into the bladder: no calculus could be detected in any part of the urinary apparatus. The walls of the abscess were constituted by all the organs in contact with the purulent collection, and by the peritoneal lining of the products of the inflammation. The adhesions between the walls of the abscess and the walls of the abdomen, which had been established by the action of the caustic, were unruptured. Peritonitis certainly existed: but it was impossible to discover any communication between the abscess and the peritoneal cavity.

The case now described is a beautiful example of chronic perinephric abscess, most probably consecutive to a pyelo-nephritis which itself had had calculous inflammation as its cause.

I do not think it necessary, Gentlemen, to dwell at any length upon the differential diagnosis of perinephric abscess, hydronephrosis, and cancer of the kidney. In the two latter diseases, although there is a tumor situated in the lumbar and abdominal regions, the progress of both affections is essentially chronic, and neither are accompanied by fever. There is fluctuation in hydronephrosis, but the lumpy state of the kidney may be sometimes detected. Though pain be a symptom in cancer of the kidney, the hardness of the cancerous tumor, and the frequent hæmaturia, will enable you to avoid any error in diagnosis.

I have already told you how to distinguish tumors of the liver from tumors of the right kidney by palpation performed during very deep inspiration; the liver moves, moving with it the tumors seated in its parenchyma, while, during similar inspiratory movements, renal tumors remain immovable. Tumors of the spleen project in so marked a manner, that it is hardly possible, even when they are very large, to mistake them for lumbar tumors. Perityphilitis, fæcal tumors of the large intestine, and fæcal abscesses can only, I think, occasion temporary mistakes: indeed, inflammation of the cæcum or appendix vermiformis, terminating in abscess of the iliac fossa or true pelvis, has so limited a seat, that error is impossible, unless it be in cases of latent perityphilitis, and in cases in which the inflammation by propagating itself to the iliac fossa and lumbar region slowly induces signs of perinephritis: in these cases, the abscess will not only yield pus having a stercoral smell, but will likewise emit a certain quantity of intestinal gases. Fæcal tumors of the large intestine have their seat in the ascending or descending colon, or they are formed as the result of long atony of the bowel: by palpation, a certain degree of softness can generally be recognised disclosing their nature: moreover, as they are removed by purgatives, all diagnostic hesitation is thus terminated.

I do not require to dwell on the diagnosis of the complications of perinephric abscess: it will be sufficient to remind you, that these abscesses may terminate by extending from above downwards, or from below upwards: in the former case, if they burrow towards the iliac fossa and true pelvis, they may cause psoïtis, and may open spontaneously into the bladder or vagina: in the latter case, they may give rise to diaphragmitis, pleurisy, or pneumonia. In conclusion, I advise you, whenever you have to diagnose the nature of a lumbar tumor to bear in mind that, at the same point where a deep-seated lumbar abscess is prominent under the skin, we may have that form of intestinal hernia with which Jean-Louis Petit has connected his name. Lately, a mistake was very nearly committed in a case of this kind, had not the surgeon before proceeding to open the supposed abscess, endeavoured to reduce the tumor.

Were it not that physicians are more frequently consulted than surgeons regarding lumbar pains, I should not have said so much about perinephric abscesses: it was important that I should give you a detailed description of deep-seated renal abscesses, sufficient to enable you in their beginning to suspect their existence, foresee their progress, and discover their etiology. I have specially brought under your notice the cases of perinephric abscesses on account of the importance of determining from the first, whether an abscess be primary, or whether it be secondary to lesion of the kidney. In the former case, the prognosis will be nearly always favorable, particularly if you early recognise the pus, and evacuate it from the lumbar region. But the prognosis is very unfavorable if the perinephric inflammation has been allowed, by temporising treatment, to advance to the iliac fossa or the diaphragm. The prognosis is also very grave, when the perinephric abscess is the sequel of calculous pyelonephritis. There are cases which show that renal calculi may, after a period of varying duration, make a way out for themselves by determining an opening in the abscess: in such cases, urinary fistulæ may remain for many years, which must not be closed, lest by so doing, the patient be exposed to the risk of new inflammatory attacks.

I have told you, Gentlemen, why perinephric abscesses are as much within the domain of the physician as of the surgeon. It is therefore my duty to give to all of you, but particularly to those of you who will have to practise both medicine and surgery, the results of my experience in the treatment of perinephric phlegmons and abscesses.

You have seen that a phlegmon may terminate in resolution. When the affection is incipient, you must endeavour to obtain that result. First of all, use every effort to calm the pain by frictions with the preparations of opium and belladonna, or by subcutaneous injection of solutions of atropia or morphia. Cupping, and large flying blisters, may be applied with benefit to the seat of pain. The bowels must at the same time be kept open by the daily use of saline purgatives and injections. Purgatives have a twofold beneficial action in these cases: they remove the constipation, and prevent the pain which would be caused by straining at stool. They also promote the resolution of the phlegmon by their antiphlogistic action.

Should these various measures fail to arrest the progress of the inflammation, and should an increase of fever with frequent rigors show that the phlegmon is suppurating, the utmost care must be taken to recognise, as soon as possible, the physical signs of suppuration. You will soon detect a doughiness in the whole of the affected region; and pressure with the hand, or the slightest movement of the body, will increase the pain. The tumor will soon become more prominent in the lumbar region; and though there may be no redness observable in that situation, you will perceive a local œdema which will convince you of the presence of pus. You will then be able to detect deep-seated fluctuation, which will be rendered more manifest by your grasping the tumor in both hands and submitting it to a brusque concussion stroke. There must then be no hesitation as to evacuating the purulent fluid. Three methods of accomplishing that object are available.

Chopart, and recently Drs. Denonvilliers, and Gueneau de Mussy, have applied caustics, so as to produce adhesions and avoid peritonitis and hemorrhage. There are numerous methods of carrying out this practice. The application of the Vienna caustic paste, repeated once or oftener to the same place, may suffice; for the natural process of eliminating the eschar, provided it be deep, will terminate by making an opening into the abscess: the pus will then flow out slowly; and in some fortunate cases, the abscess will be entirely evacuated, deep cicatrisation taking place at the same time that superficial cicatrisation is accomplished on the falling off of the eschar. Though this method of proceeding has great advantages, it has often, in my opinion, the great draw-back of being very slow, and so affording time for the abscess to extend to the iliac fossa or diaphragm, or open into the intestine. The advocates of the method are quite aware of this objection: MM. Denonvilliers and Gueneau de Mussy have, on the second or third day after applying the caustic, cut out the eschar to get at the abscess. This mixed method——cauterisation and incision—has the advantage of diminishing the thickness of the tissues which have to be traversed by the bistoury, and causing the formation of adhesions between the superimposed tissues. It has the disadvantage of risking hemorrhage, by the bistoury cutting into deep-seated vessels which had not come under the influence of the caustic. I prefer to make the incision in the first instance, taking care, however, to cut layer by layer, and to ligature all the arteries divided by the bistoury. If this principle be adopted, it is of little consequence whether the incision be longitudinal or transverse. However, when the thickness of the lumbar wall renders it difficult to tie the deep lying arteries, it will be more prudent, after cutting through the superficial parts, to separate the deep parts by tearing them with a canulated sound. The risk of dividing an artery will thus be obviated. The incision through the superficial, must be larger than through the deep, parts, so that the pus be not detained in the wound to detach the tissues and burrow between the subcutaneous aponeuroses and muscles. A large pledget of charpie ought to be inserted in the wound so as to reach into the abscess. Any venous or capillary hæmorrhage which may arise from the lips of the wound will be easily arrested by the application of some pieces of agaric. When the patient's general state is satisfactory, and when the perinephric abscess is primary, the walls of the abscess will soon contract on themselves, and in a fortnight or three weeks from the date at which the opening was made, complete cicatrisation—deep and superficial—may be expected. Sometimes, however, the suppuration continues for a longer period, which may depend on a difficulty in the flow of the pus, or on some special condition of the walls of the abscess. In the former case, the deep opening must be enlarged; and in the latter case, detergent injections must be thrown into the cavity of the abscess: the injections, which ought to be repeated at each morning and evening dressing, may consist of tincture of iodine diluted with two or three times its weight of tepid water. Incision has the advantage of giving an immediate outlet to a great quantity of pus, and allowing a digital exploration to be made of the kidney, so as to ascertain its position and discover whether it be or be not diseased. It is unnecessary to add that, when the finger detects the presence of calculi imprisoned in the kidney, nothing ought to be done to favor cicatrisation of the surgical incision: on the contrary, a fistula ought to be maintained to enable urine and pus to find an exit.

The method of drainage, to which M. Chassaignac has several times successfully had recourse for the evacuation of deep-seated lumbar abscesses, allows both a continuous flow of pus and the gradual contraction of the cavity and the abscess. It also lessens the risks of hemorrhage, and does not appear to favor purulent infection; but it has the disadvantage of hardly allowing complete exploration of the abscess and the kidney. Moreover, the presence of the tubes frequently maintains a purulent discharge for a long period.

LECTURE XCVII.

PERIHYSTERIC ABSCESS.

Perihysteric Abscess, including Phlegmon of the Broad Ligament and Pelvi-peritonitis or Female Orchitis.—Etiology.—Symptoms and Duration of Pelvi-peritonitis.—Perihysteric Tumors.—Spontaneous Opening of the Abscesses into the Intestine, Bladder, and Vagina.—Complications.—Diagnosis of Perihysteric Abscesses.—Preventive Treatment of Perihysteric Abscesses.—Active Intervention only proper in the Iliac Abscesses.

GENTLEMEN:—You know my dislike to neologisms, and the pain it gives me to become the author of a new word. I have a respect for old names; yet I avow, that I feel it very distasteful to employ barbarous terms such as those which result from the alliance of a Greek preposition with a Latin substantive. Hence my objection to the word *periuterine*, employed to designate different affections which have their seat around or in the neighbourhood of the uterus. It is a hybrid, badly coined word, for which I propose, to substitute *perihysteric*, from περι and ὑστερα. It has the advantage of being composed of two Greek words. I have, however, so little paternal partiality for the term, that if you prefer the word *circumuterine*, recently introduced into medical nomenclature, I am quite willing to accept it.

Having disburdened myself on that preliminary point, I now enter at once into the subject. In the study of perihysteric abscess upon which we are now entering together, I propose that we do not confine our attention to abscesses situated around the uterus, but that we likewise consider those which have secondarily invaded the iliac fossæ and the sacro-iliac symphysis. It is necessary thus to extend the limits of our study, on account of the impossibility to be always able to determine with precision the starting point, the primary seat of perihysteric affections.

Gentlemen, I take it for granted, that you recollect the principal details of the anatomical typography of the female pelvis. Allow me, however, to remind you of the great, special features in its anatomy, because unless you possess that knowledge, it will be impossible for you to understand what I have to say on the progress of perihysteric abscesses.

The cavity of the pelvis, which contains and protects the uterus and its annexes, may be divided into two regions—the anterior, and the posterior. The uterus and the broad ligaments constitute the boundary between these two regions. The uterus, the normal axis of which is the axis of the true pelvis, is suspended in the pelvic cavity, and maintained in its normal relations by the broad ligaments and the *retro-uterine* and *ante-uterine* ligaments. These last named ligaments, you are aware, are formed by folds of the peritoneum, which proceed from the rectum and bladder, to be inserted in the inferior lateral portion of the body of the uterus. At these points, the serous membrane is strengthened by fibres of fibrous and muscular tissues. The broad ligaments have the same fibro-muscular structure as the ante-uterine and retro-uterine ligaments, but they are much wider, and have a quadrilateral form: they present, in apposition, two layers of peritoneum, containing in their upper margin the Fallopian tubes, the round ligament of the uterus, and the ovarian ligament. In that situation in particular, there are numerous fibres of connective and muscular tissue: there, also, converge the utero-ovarian vessels. The inferior margin of the broad ligament is inserted in the floor of the pelvis. The internal or uterine margin is a continuation of the folds of peritoneum which cover the anterior and posterior surfaces of the uterus. Between them, at the point at which they are inserted into the uterus, there is a space, the dimensions of which vary with the degree in which the organ is full or empty; but they are always so firmly adherent to the anterior and posterior surfaces of the uterus by cellular tissue, that it is impossible, by the most patient dissection, to detach the peritoneum at these points, without tearing it. This anatomical fact is very important; because it establishes the impossibility of the peritoneum in that situation being separated by circum-uterine suppurative inflammation. The external margin of the broad ligament is the widest: its extreme limits are the iliac fossa and the floor of the pelvis: consequently, by means of the cellular tissue which joins the two peritoneal folds, it is in immediate relation with the subperitoneal cellular tissue of the iliac fossa and pelvic cavity. These insertions, while they keep the empty uterus in its position and normal relations, are not so rigid as not to yield when the organ is increased in size, as it is in certain pathological states, or in cases of tumor of the broad ligament. If you keep in view these anatomical relations, you will easily understand my description of tumors situated before, behind, or in the broad ligament. Again, by recollecting the relations of the round ligament and ovary with the cellular tissue of the iliac fossa and cavity of the pelvis, you will be easily able to follow the advance of purulent collections in these different situations.

In a previous lecture, when treating of perinephric abscess, I explained to you how abscesses originating in the circum-renal cellular tissue might burrow in that tissue, so as to reach the true pelvis and open into the bladder or vagina. When the pus flows into the vagina, we can sometimes see, by the aid of the speculum, the fistula by which it is discharged: before the spontaneous rupture of the abscess, a tumor of greater or less volume on the side of the uterus could, by digital exploration, be recognised, projecting into the vagina. I remind you of these facts, because they show beyond the possibility of doubt, that the cellular tissue of the pelvis may be the seat of purulent collections without the existence of any peritoneal inflammation. Here, let me repeat, that it is exceedingly unusual for such abscesses to open spontaneously into the peritoneum. You are thus enabled to anticipate my statement, to the effect, that real abscesses may be formed in the perihysteric cellular tissue, and that they are not always attributable to pelvi-peritonitis.

However, to be convinced of the existence of abscesses in the cellular tissue of the pelvis, it is well to recollect certain facts in pathological anatomy, which can seldom be verified, as the patients do not generally die till after the inflammation has gone on for several weeks or months, and has extended to the peritoneum, rendering the anatomical demonstration quite impossible.

Most of you, no doubt, remember the autopsy of a young woman who died in Saint-Bernard's ward, consequent upon purulent infection supervening a fortnight after delivery. I was anxious to discover the seat of the purulent infection: no morbid change was visible in the uterine veins, but the walls of the vagina were covered with pustules situated in a vascular network. Moreover, the cellular tissue lining the vagina presented all the characters of inflammation: that is to say, the layers of tissue were engorged with a yellow serosity, in some places having a purulent appearance; while, at the same time, the tissue had acquired a special hardness. The phlegmon extended to the sides of the pelvis. In difficult labours, it is not unusual to observe contusions of the soft parts of the pelvis leading to engorgement of the cellular tissue of the contused parts. The engorgements may terminate in resolution; but they may sometimes also become the origin of pelvic abscesses projecting around the uterus, the peritoneum not being necessarily implicated in the inflammatory action. In Dr. Dumontpallier's inaugural thesis, you will find the case of a young woman who died on the sixth day after delivery from a very acute attack of non-purulent traumatic peritonitis. The cellular tissue of the left iliac fossa contained pus; but it was particularly in the true pelvis that the most serious morbid changes were found. The whole of the cellular tissue attaching the different organs to the osseous parietes was infiltrated with purulent serosity, and presented in some places, particularly on the left side, layers of sanguineous deposit, the manifest evidence of great contusion. It is probable, that had this patient not succumbed under the severe peritonitis, she would ultimately have presented all the signs of iliac and pelvic abscess.

It appears then, that contusion during labour may be the origin of pelvic phlegmons, which, becoming developed and going on to suppuration, produce a perihysteric abscess. This cause of pelvic phlegmon is much less common than inflammation of the uterus and its appendages. When there is suppurative inflammation of the placental surface, uterine phlebitis and lymphangitis are, as you know, very frequently observed. When, at the autopsy, you look for pus in these vessels, you know that the chosen place for it is in the veins at the margin of the uterus: an incision made in these vessels, the true confluents of the uterine veins, nearly always discloses small abscesses situated in the venous tissue itself. Moreover, around these veins, the cellular tissue of the broad ligament is œdematous; and if the patients do not die from purulent infection, it is because there has been adhesive phlebitis further down the stream than the purulent collection: the intra-venous abscesses are often the origin of abscesses of the broad ligament. A similar remark is applicable to suppurative lymphangitis. In these cases, the peritoneum does not participate in the inflammatory action; and the abscess, if not absorbed, will tend towards the depending parts, or the inflammatian may extend to the iliac fossa of the same side.

In the former case, the abscess, in the form of a tumor, will have its seat in the lateral parts of the uterus; and, sooner or later, will project into the vagina. You then have an abscess of the broad ligament, that is to say, a suppurative inflammation of the cellular tissue which unites the two peritoneal folds, between which lie the inflamed vessels, veins or lymphatics, the starting point of the phlegmon.

Not only delivery, but any traumatic condition of the mucous membrane of the uterus, may occasion perihysteric abscesses. For example, they have been seen after abortion, and after surgical operations, such as scraping the uterus by Récamier's instrument. The actual cautery, and even the application of the nitrate of silver, have sometimes given rise to similar phlegmons. Perhaps a similar result has been caused by excessive coitus. I am inclined, however, to think that this kind of excess more frequently produces pelvi-peritonitis. Though primary inflammation of the veins or lymphatic vessels is the most frequent cause of an abscess, conditions necessary for its formation are a solution of continuity and inflammation of the mucous membrane of the uterus. These lesions exist after delivery, abortion, and the application of caustic or cautery: but I can hardly understand their being produced by excess of coitus, unless, indeed, uterine catarrh or ulceration of the cervix uteri previously existed.

Be that as it may, once the abscess is formed in the thickness of the broad ligament, what is the usual course of its progress? Most frequently, as I have said, it will project at the side of the uterus, and open into the vagina or bladder. It usually occurs on one side of the uterus only, and does not project either behind or in front of that organ. When an abscess is situated behind or in front of the uterus, the conclusion is that the pus is in the peritoneal cavity; or —to make the same statement in other terms—the abscess is due to pelvi-peritonitis. In point of fact, an abscess cannot invade an ante-uterine or a retro-uterine cellular tissue, inasmuch as neither exist!

When there is a real perihysteric abscess, that is to say, when the purulent collection is situated around the uterus, in the sub-peritoneal cellular tissue, and not in the cavity of the peritoneum, the pus sometimes reaches the iliac fossa, giving rise to the signs of abscess in that fossa—signs which vary according as the abscess is superficial or deep. It is generally superficial, and uncomplicated with psoïtis. Suppurative inflammation, moreover, must, it is evident, follow the cellular tissue to reach the iliac fossa.

When an iliac abscess is formed, whether consequent upon propagation of the inflammatory process from the broad ligament, or on inflammation primarily seated in the iliac fossa, pain is felt in the place where the abscess is situated. By moderate palpation, doughiness of the region is detected; and there is sometimes slight œdema of the abdominal walls. When the abscess is situated in the right iliac fossa, it may open into the cæcum, but more frequently, it points immediately above the crural arch. At other times, if prompt issue be not given to the pus, it may, following the vessels, work its way to the triangle of Scarpa, or reach the trochanter minor and coxo-femoral articulation.

When the last-described course is taken, the iliac abscess is generally deep-seated, sub-aponeurotic, and complicated with psoïtis. These deep-seated iliac phlegmons almost always terminate in death, the patients sinking from exhaustion consequent upon the suppuration. I could by narrating many examples prove these cases to be as serious as I have now stated. I will confine myself, however, to the description of one case, which presents many complications of great clinical interest; and shows the amount of damage produced by purulent collections of this kind. The patient, thirty-five years of age, was delivered on August 30th, 1861, at the Hôpital des Cliniques, where she remained till the 18th September, experiencing pain in the hypogastric region. On leaving the hospital, she kept her bed at her own home till the 5th October, the date at which she was admitted to the Hôtel Dieu, bed 5, Saint-Bernard's ward. I then ascertained that there was a phlegmon of the right broad ligament. Soon, there was an abscess; and the inflammation invaded the right iliac fossa. Early in November, the urine was observed to deposit a large quantity of pus. Very probably, a fistulous communication had been established between the abscess of the broad ligament and the bladder.

The inflammation of the iliac fossa nevertheless continued to advance; and about the middle of November, a tumor could be readily recognised, tending towards the crural arch. At the same time, very acute pains supervened in the iliac region, and throughout the whole of the inferior extremity: extreme pain was caused by the least pressure on the tumor, or by the slightest movement of the limb. The pain was constant: and paroxysmal exacerbations extorted cries from the patient.

The iliac tumor pointed immediately above the crural arch, where fluctuation could be distinctly felt. As, at intervals, the urine had a purulent deposit, it was thought that the iliac abscess would gradually empty itself by the bladder. It did not, however, so happen. The pain continued very acute, with paroxysmal exacerbations. For several weeks, the inferior extremity had assumed the position described by authors as belonging to psoïtis: that is to say, the thigh was slightly flexed on the pelvis, the leg was flexed on the thigh, and the whole limb (propped up by pillows) was to a small extent rotated outwards. The fever was constant; the pulse was small and frequent, becoming doubled in frequency in the evening: profuse nocturnal sweats occurred. As the patient was becoming weaker, I requested M. Alphonse Robert to open the abscess. The incision was made above the crural arch, where the tumor pointed, four or five centimeters from the anterior superior spine of the ilium. Hardly had the skin been divided, when there was a copious gush of pus, at first greenish, fœtid, very grumous, and afterwards sanguinolent. On the morning after the operation, the 10th December, the state of the patient was relatively better: she had slept a little during the night. There was no fœtor in the mingled pus and blood which came from the incision. It was desirable to increase the patient's strength by appropriate diet; but on the 12th December, the severity of the fever was doubled, and the mucous membrane of the mouth was covered with aphthous patches: deglutition became painful, difficult, and at last impossible. The voice became snivelling and very feeble, the breathing oppressed, and the bronchial passages engorged. The patient died on the 13th December, four days after the opening of the abscess. In the last days of her life, she presented none of the symptoms of purulent infection.

An autopsy was made on the 15th December. The intestines were carefully removed *en masse*, to afford greater facility for studying the relations of the iliac abscess. A very large sub-aponeurotic abscess was then seen in the iliac fossa, in the pus of which were bathed the psoas magnus muscle, the iliac vessels, and the crural nerve. There was a very considerable quantity of pus in the cavity of the abscess, which was circumscribed by the iliac aponeurosis and hardened cellular tissue. The edge of the ilium formed the superior boundary of the abscess: inferiorly, was perceived the incision made by the surgeon just above the crural arch. Below the crural arch, the abscess had two prolongations, one of which followed the psoas muscle to its insertion in the trochanter minor, and the other followed the crural nerve. The purulent train which followed the tendon of the psoas had invaded the coxo-femoral articulation, which was open and full of pus. The head of the femur was denuded of its cartilage. The train of pus which had taken the course of the crural nerve stopped four or five centimeters below the femoral arch. The crural nerve was bathed in pus, and its neurilema was of a blackish color. The femoral vessels were free in the middle of the abscess: they were enclosed in a sheath of indurated cellular tissue: there was no noteworthy change visible in the artery: the vein contained newly formed cruoric clots, which were non-adherent, and had never embarrassed the venous circulation. No morbid change was visible in the vena cava inferior.

As I have remarked, the coxo-femoral articulation was very much altered by the suppuration: the same statement may be made in respect of the right sacro-iliac symphysis, which was open and full of pus, and the articulating surfaces of which were evidently in a state of inflammation. The abscess of the broad ligament, which had been very probably the starting point of all the pathological changes, had no direct communication with the iliac abscess. The peritoneal coats of the broad ligament were much thickened, and the uterus was almost glued to the right wall of the pelvis by the contraction which had taken place in the diseased tissues subsequent to the evacuation of the pus by the vesical fistula.

During life, I had been led to the conclusion that there was a vesical fistula; and at the autopsy, I had searched for the communication between the broad ligament and the cavity of the bladder. An opening having been made in the bladder, in its anterior superior part, a vesical fistula was observed, which communicated with the primary abscess of the broad ligament. This fistula was situated in the right lateral portion of the fundus of the bladder, three or four centimeters behind the orifice of the right ureter.

The uterus, vagina, and rectum, on careful examination, were found not to communicate with the abscess, and to present no morbid change. The pus was in immediate contact with the psoas magnus; but there was no morbid change in its fibres, except that the superficial fibres had a greenish colour, due to contact with the purulent fluid.

The lungs were engorged, but presented no traces of inflammation. Both apices contained some small masses of softened tubercle. The lungs and liver were both free from any trace of metastatic abscesses. On some parts of the surface of the liver, there were small irregularly shaped yellowish spots of the size of a twenty centime piece, due to the presence of numerous fat-globules in the hepatic cells. The spleen was small, and not softened. The kidneys were normal in colour, form, and texture.

The pathological physiology of these cases appears to me exceedingly interesting. Here, a recently delivered woman had abscess of the broad ligament, and the inflammation had extended by continuity of the cellular tissue of the true pelvis and iliac fossa. The abscess of the broad ligament had emptied itself into the bladder, whilst the sub-aponeurotic abscess made great ravages, penetrating into the sacro-iliac symphysis, dissecting the crural nerve, pointing above the crural arch, after having detached the superficial fascia of the anterior abdominal wall: it was at that point that the surgeon introduced his bistoury. The abscess had burrowed below the crural arch, following in one direction the crural nerve, and in the other, the psoas muscle to its trochanterian insertion. It advanced to the coxofemoral articulation; and denuded the head of the femur of its cartilage.

The connexions formed between the uterus and broad ligament and fundus of the bladder, by cellular tissue, explained the opening of the pelvic abscess into the bladder. The progress of the iliac abscess, and its tendency to burrow towards the regions which I have mentioned, may be foreseen: but it is not less remarkable to see the ravages of the inflammation extend to the sacro-iliac and coxofemoral articulations.

The grave articular changes would be sufficient in themselves to explain the pains which the patient suffered: every movement occasioned frightful torture: and though it was not forgotten that the crural nerve had been dissected by the pus, that many of its branches detached by the inflammation of the nervous bundles were swimming in it, there was found in the articular mischief an explanation of the acute pains which darted through the inferior extremity with that paroxysmal character so remarkable in lesions of the nerves.

Here, Gentlemen, allow me to make a short digression, touching the value of the lesion of the crural nerve in explaining the symptoms of psoïtis. This lesion seems to me to explain the symptoms which we have observed, and which are generally attributed to psoïtis. In our patient, the psoas magnus muscle had undergone no pathological change. Is it not, therefore, reasonable to suppose, that the symptoms attributed to psoïtis do not belong to any lesion of the muscle, but to lesion of the crural nerve?

In proof of the value of this remark, it is sufficient, on the one hand, to find cases of inflammation of the psoas muscle without symptoms of psoïtis; and on the other, to recognise these symptoms in cases in which the crural nerve has been seriously implicated in the process of inflammation. By a singular chance, I, next day, met with these latter conditions. The deceased was a young man who had just succumbed from typhoid fever in Dr. Horteloup's wards. Dr. Lefeuvre, then *interne* of the service, invited me to verify the existence of an enormous abscess in the substance of the psoas muscle. The pus was thick and mingled with blood. The muscular fibre was partially disorganised: some portions of it, when examined under the microscope, presented the appearance of pale longitudinal striæ, and the transverse striæ were only visible in some places: the muscular sheath contained a quantity of small shining fat-globules. The nerve-bundle constituting the crural nerve had not been involved in the inflammation. The limb was extended, and parallel with that of the opposite side.

Dr. Lefeuvre informed me, that the patient had never complained of pain, nor of any symptom which could lead to the conclusion that there was psoïtis. You see, therefore, that in this case, there was great lesion of the psoas muscle, without any symptoms which could be attributed to psoïtis.

I thought that the psoas abscess was probably a metastatic abscess resulting from purulent infection, the anatomical cause of which was probably ulceration of the glands of Peyer. Purulent infection is an unusual sequel of typhoid fever: here, however, was a case in which it had occurred, as was proved by the presence of numerous metastatic abscesses in both lungs. Having shown that this case takes its place with other cases of purulent infection, I now return to the principal fact, viz., the absence of the symptoms of psoïtis in a case of undoubted suppuration of the psoas muscle.

In this case, then, there was psoïtis properly so called, that is to say, inflammation of the inter-fibril cellular tissue, along with disorganisation of the muscular fibre, yet there had been none of the symptoms attributable to psoïtis, and—let me call your special attention to the fact—there was no lesion of the crural nerve.

From the conjoint consideration of these two cases, we may, I think, draw the following conclusions:—

1. That the symptoms of psoïtis specially depend on lesion of the crural nerve.

2. That in certain cases of psoïtis, a great pathological change may take place in the psoas muscle, there being nevertheless an absence of symptoms attributable to psoïtis.

3. That inflammation of the psoas muscle and inflammations of the iliac fossa, may often give rise to the alleged characteristic symptoms of psoïtis when the crural nerve has been secondarily invaded by the surrounding inflammation. [In justice to Professor Grisolle, I must mention, that in his remarkable memoir on iliac abscesses, he attributed a great share to lesion of the lumbar plexus and crural nerve, in explaining the pains which accompany iliac phlegmons and abscesses.[1]]

We have seen, Gentlemen, that deep-seated iliac abscesses may produce grave alterations in the coxo-femoral and sacro-iliac articulations: the existence of these lesions is generally unknown till disclosed at the autopsy. In a subsequent lecture, when discussing relaxation of the symphysis of the pelvis, before and after delivery, I will describe two cases in which there was suppuration of the sacro-iliac symphysis. In neither of these cases, was the articular inflammation the consequence of an intra-pelvic abscess.

In the case which I have just related an abscess of the broad ligament seemed to have been the origin of all the symptoms. However, abscesses of the broad ligament are not so frequent as some years ago they were said to be. Dr. Bernutz, and his lamented fellow-worker, Goupil, in their memoir published in 1857,[2] endeavoured to show that pelvi-peritonitis is very common. They showed that in many cases in which the physician had made out all the signs of acute or chronic plegmon of the broad ligament, it had been demonstrated at the autopsy, that the cellular tissue of that ligament had not been involved in the inflammation, and that the sole seat of the products of inflammation had been the peritoneum of the true pelvis. Hence these physicians, after collecting a great number of cases came to consider the pelvic peritoneum in the female as analogous to the tunica vaginalis in the male subject.

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[1] Grisolle:—*Archives Générales de Médecine.* Third Series (1839): Vol. IV.

[2] *Archives Générales de Médecine* [March and April, 1857]; and Clinique Médicale sur les Maladies des Femmes. T. II, Paris: 1862.

Orchitis, or, to speak more correctly, vaginalitis, ought, therefore, to have pelvi-peritonitis as its corresponding affection in the female. Just as lesions of the urethra, prostate gland, and testicle often originate orchitis, so (it is argued) must lesions of the vagina, uterus, Fallopian tubes, and ovaries, often produce pelvic peritonitis. I am willing to accept this ingenious interpretation of facts, and to recognise with MM. Bernutz and Goupil the existence of blenorrhagic, traumatic, tuberculous, and other kinds of pelvi-peritonitis. This classification has the advantage of obliging the clinician to carry back his investigation to the cause of the inflammation of the pelvic peritoneum. Science has long been in possession of the histories of numerous cases of these different kinds of pelvi-peritonitis; and practitioners in their daily practice are called upon to verify the symptoms of a localised peritonitis in women affected with vaginitis and acute or chronic inflammation of the uterus. All of you must have present to your minds some necroscopic examination in which inflammation of the Fallopian tubes and ovaries was seen to have afforded the starting point for a more or less extensive peritonitis.

It is particularly as a sequel to normal or artificially induced labour, that we meet with inflammation of the tube and ovary. In these cases, the peritoneum surrounding or adjoining the tubes and ovaries soon takes on the inflammatory action. Pain and swelling localised at some particular part of the pelvis, enable us to recognise the seat and nature of the lesion. Adhesions form between the affected parts; and if the peritonitis do not become general, the patients may recover after a certain time, retaining the cellular adhesions, which will make their presence known by occasioning pain, particularly at the menstrual epochs. You will often observe, that these attacks of limited peritonitis are connected with some acute lesion of the ovary. The frequent recurrence of the inflammation may occasion numerous adhesions between the pelvic organs, whence may arise errors in diagnosis, if great care be not taken to mark well the progress of the engorgements, and observe the signs furnished by palpation and percussion. These numerous adhesions, uniting in the form of a tumour of the uterine annexes, to portions of the intestinal mass, sometimes extensive, lead to serious consequences, they impede the performance of the functions of organs, and produce marasmus.

I have not often observed pelvi-peritonitis as a consequence of inflammation of the vagina; but every year, I frequently meet with peritonitis consequent upon metritis. Bear in mind the pains in the lower part of the abdomen complained of by some of our patients—pains accompanied by nausea, vomiting, fever, and dysuria—in the evening of the day or on the day following that on which we have applied caustic to the neck of the uterus. These pelvi-peritonitic symptoms generally subside in a few days: the inflammation, however, may extend to a great part of the peritoneum, when the patients may be carried off by very acute peritonitis.

Sometimes, mortal peritonitis is occasioned by leaving an instrument in the cavity of the uterus. In the discussion on stem-pessaries, which took place in the Academy of Medicine, several such cases were mentioned. The temporary introduction of the hysterometer has occasioned very alarming peritoneal symptoms. From these statements, there can be no doubt that an apparently slight uterine lesion may give rise to very acute general peritonitis: and, therefore, of course, we may assume, that acute metritis will much more readily occasion peritonitis limited to the true pelvis.

Pelvi-peritonitis, Gentlemen, is characterised by effusion of serosity into the true pelvis, and the formation of adhesions which give rise to perihysteric tumors. These tumors, variable in volume, are situated at the sides of the uterus, or in the ante-uterine and retro-uterine cul-de-sacs. Sometimes, the broad ligaments assist in developing the tumor: in such cases, the uterus seems to be firmly nailed to the middle of the tumor. When the retro-uterine cul-de-sac is the seat of the pelvi-peritonitis, the uterus is carried forwards behind the symphysis of the pubes, as happens in retro-uterine hæmatocele. When the pelvi-peritonitis is more severe on one side than the other, lateral deviation of the uterus will be produced. The different deviations of the uterus are only of secondary importance. The great fact which you require to bear in mind is, that these peritoneal inflammations, which often become encysted abscesses, have a tendency, whether they be acute or chronic, to evacuate themselves by the rectum, vagina or bladder, so that in a single day may disappear a great part of the tumor which occupied the true pelvis.

Pelvi-peritonitis is sometimes very protracted, and occurs in paroxysms. Some years ago, a surgeon who was treating one of my patients for a uterine affection of old standing, proposed to employ Récamier's scraper [*curette de Récamier*] to destroy fungous excrescences on the mucous membrane of the uterus. It is always a serious proceeding, Gentlemen, to introduce a foreign body into the uterus, particularly when that is done with a view to remove diseased portions of the mucous membrane: there is always a risk of its producing metritis and its consequences. Under the circumstances, therefore, I proposed that another surgeon, a Professor of the Faculty, should meet us in consultation. My accomplished colleague realising the great responsibility which he was assuming, wished to examine the affected organs with great care, so as to be able to state his reasons for the advice he gave. The digital examination occupied a long time; and was, the patient said, very painful. Possibly, there already existed latent chronic pelvi-peritonitis: it is important to note that from the day of our consultation, the patient felt almost constant pain in the lower part of the abdomen—pains which several times within the month assumed intense severity. For three years, our patient, obliged to keep her bed or lie on a sofa, passed a large quantity of pus by the anus. I need not say, that on all occasions, during this long period, the necessary digital examinations were made with the utmost caution. Several times, I had an opportunity of ascertaining that there was a doughy tumidity [*empâtement*] within the pelvis, particularly in the retro-uterine region. Finally, in this patient, coagulations formed in the pelvic veins. One day, when the acuteness of the pains had rendered an examination necessary, the patient was suddenly seized, during the examination, with the symptoms of pulmonary embolism. The symptoms gradually abated: some months later, the extent of the perihysteric engorgement diminished: no more pus flowed from the rectum. The patient may to-day be described as cured.

Very probably, this was a case of pelvi-peritonitis, and not of perihysteric phlegmon. The progress of a phlegmon is different and less protracted: pelvi-peritonitis, on the other hand, as necroscopic examinations prove, is characterised by chronicity; and when the adhesions are well established, it may, under the influence of a variety of causes, be the source of ceaseless suppuration. In saying this, I do not mean to express a positive belief that the cellular tissue lining the peritoneum remains exempt from the inflammation primarily seated in the pelvic peritoneum. In fact, I have difficulty in understanding how a chronic phlegmasia accompanied by an abscess opening into the rectum, vagina, or bladder, should always remain limited to the peritoneum: I do not believe that that is the rule, although MM. Bernutz and Goupil have given very remarkable cases in support of such a view. In reality, chronic inflammation so thoroughly invades the cellular tissue of the pelvis, Fallopian tubes, and ovaries, that, at the autopsy, it is sometimes very difficult to distinguish any trace of these organs, and impossible to say the order in which the different organs were attacked, unless account be taken of the progress of the symptoms during life.

My patient's case proves that a chronic lesion of the mucous membrane of the uterus may lead to latent pelvi-peritonitis, and that a determining cause, such as digital examination, may bring on acute symptoms. Moreover, these symptoms, become chronic, may have paroxysmal exacerbations declaring themselves by more or less purulent discharge. Again, the blood may coagulate in the pelvic veins surrounding the inflamed tissues; and clumsy or inopportune manipulations may break up the clots, and so lead to their migration to the heart and lungs. Under such circumstances as I am now referring to, you cannot be too reserved in your prognosis, nor too prudent in your examination of the affected organs.

Pelvi-peritonitis may also lead to other consequences. Not only from its long duration may the suppuration cause great wasting of the body and hectic fever, but it may likewise, though not so frequently, lead to purulent infection. Chronic pelvi-peritonitis may also, in predisposed subjects, by its long continuance, lead to tubercular affection of the lungs and peritoneum.

The peritonitis does not always remain limited to the pelvis: I have often seen it spread so widely as to comprise a great portion of the intestines in its adhesions. The extension of the peritonitis is particularly observed after delivery and abortion. Numerous autopsies show, that a portion of the small intestine sometimes becomes united in one common mass with the great omentum and the organs contained in the cavity of the true pelvis. When that is the case, and when the patients do not die under the acute symptoms, they soon grow weak. The pus, however, which occupies the inflamed masses, at last makes an issue for itself; and in this way, recovery may take place. You remember the case of the young woman who occupied bed 23, Saint-Bernard's ward. On three occasions, with some days of interval between each, she presented the signs of peritonitis, which, each time, made progress; the acute symptoms abated: the tumour became circumscribed, and discharged its pus by opening spontaneously into the vagina. Some weeks afterwards, the woman left my wards quite recovered, except that she continued to have the abdominal pains inseparable from extensive adhesions of the abdominal peritoneum.

As I have already said, inflammation of the Fallopian tubes and ovaries, so common in recently delivered women, may be the source of pelvi-peritonitis and phlegmons of the iliac fossa. But under certain circumstances, when there is no adhesion of the tube or ovary to the surrounding parts, the abscesses of these organs may open into the cavity of the peritoneum, and (as I have twice seen) give rise to rapidly mortal attacks of peritonitis.

I have now, Gentlemen, brought under your notice several cases which were phlegmon of the broad ligament, iliac abscess, pelvi-peritonitis, ovaritis, and metro-peritonitis; and you may have remarked, that some of the earliest symptoms specially suggested the particular affection: but you have also seen, that as the case advanced, the differential characteristics gradually became effaced by the mere advance which was taking place in the disease. In several of the patients, examination of the body after death showed that there was both pelvi-peritonitis, and abscesses in the pelvis, iliac fossa, or even in the renal region. You see, therefore, that it would be very difficult to give you a clinical description of each of these affections; as they have such a variety of causes embracing traumatic influence, which is the most simple, and the puerperal state, which is the most complex cause.

I am now about to try to present to you some clinical views, by which you may be enabled to distinguish certain varieties of the different affections we are now considering. It is unusual for any one of them to exist separately: they are generally accompanied by numerous lesions of the uterus, ovary, or peritoneum.

When the abscess is situated in the broad ligament, digital examination will enable you to distinguish a very decided resistance to pressure in one side of the uterus: and if you, at the same time, apply the free hand to the anterior wall of the abdomen, you may by steady gentle pressure, detect a tumor, of greater or less size, in the pelvis. The conditions under which the tumor is developed, the fever, and the pain, are very important elements of diagnosis, and justify the opinion that the phlegmon is situated in the broad ligament. The relations of the uterus to the tumor will also assist in the diagnosis; and when the patients in the beginning of the disease have had neither nausea nor vomiting, and when the seat of the pain has remained confined to the situation of the tumor, it is exceedingly probable that the inflammation has not extended to the pelvic peritoneum. It also not unfrequently happens, that the patients complain of only very slight pains in the hypogastrium; in such cases, you will find that the tumor is limited to the region of one of the broad ligaments. In these cases, digital examination by the rectum proves that the retro-uterine cul-de-sac does not contain any product of inflammation. If no peritoneal complication supervene, the abscess will open into the vagina or bladder.

Iliac phlegmons are sometimes the consequence of the propagation of the inflammation to the broad ligaments: at other times, particularly after delivery, they originate primarily in the iliac fossa. Whatever may have been the conditions under which they originated, they can always be easily recognised by the pain and doughy tumidity of the affected region. Moreover, the progress of the inflammation will soon supply the elements necessary for diagnosis, and show these abscesses to be either superficial or aponeurotic.

When the abscesses are superficial, the pains are limited to the affected region; but if opening the abscesses be delayed, they become sub-aponeurotic: the pains will then extend to the thigh following the course of the crural nerve: and the abscesses will very soon point above the crural arch, or burrow deep down in the thigh. You have seen the destruction to the coxo-femoral articulation, and sometimes though not so frequently to the sacro-iliac articulations, induced by these abscesses.

The prognosis is favorable, when the abscess is limited to the broad ligament. On the contrary, if it be situated in the iliac fossa, there is reason to fear its becoming sub-aponeurotic, and endangering the life of the patient.

I need hardly say, that when the abscess is situated within the cavity of the peritoneum, the diagnosis is simplified by the previous peritonitis. When the abscess is large, its walls are formed by intestine, great omentum, uterus, ovary, ovarian annexes, and bladder. A tumor is thus formed, situated in the true pelvis, and also, occupying to a considerable extent the hypogastrium. In these regions, a peculiar doughiness is detected, in the midst of which the experienced hand can sometimes recognise, in thin subjects, by palpation, a non-homogeneous resistance. On percussion, different degrees of dulness are perceived, showing that a portion of intestine is involved in the tumor. It is found, by digital examination, that a change has taken place in the relations of the uterus—that it has lost its mobility, and is nailed, as it were, to the middle of the tumor. At the bottom of the vagina, and in the cavity of the pelvis, the finger feels a swelling, which extends to the sides of the pelvis and to the retro-uterine cul-de-sac. When such conditions exist, it is certain that the peritoneum is the seat of inflammation.

But it must not be supposed that pelvi-peritonitis, in whatever cause it originates, has always such precise diagnostic signs. It often becomes developed as a latent affection after delivery and abortion, or arises from inflammation of the uterus and vagina. We then find, that though the patients experience some pains, they neither have vomiting nor nausea. The ordinary assemblage of symptoms of peritonitis is absent, and I confess that it is very difficult to assign to this form of pelvi-peritonitis any distinctive characters. Modern works, however, and particularly the memoir of Goupil and Bernutz, have so completely established the great frequency of pelvi-peritonitis, that you must always endeavour to detect, in the antecedents of the patients, a cause for the inflammation of the peritoneum.

For the present, I avoid introducing the more difficult cases into the discussion; and confine myself to a consideration of the morbid characters which seem to explain the more common facts of pelvi-peritonitis. When patients suffer from pain in the hypogastrium as a sequel of sexual excess, or as an accompaniment of gonorrhœa or metritis, the possible existence of pelvi-peritonitis immediately suggests itself. If it exist, palpation of the hypogastrium will produce pain, and digital examination by the vagina, if pain has existed for some previous days, will detect a tumor occupying the perihysteric region. The pains then occur in paroxysms, while the tumor becomes more and more appreciable to the means of investigation employed. These abscesses of the pelvic peritoneum, sooner or later, and at intervals, open into the vagina, bladder, or rectum.

The prognosis in pelvi-peritonitis varies with the extent and cause of the inflammation. When it involves a portion of the intestine and the annexes of the uterus, the case is one of great gravity, because hectic fever will soon set in. Orchitis in the female, on the contrary, when limited to the pelvis, and not interfering with the functions of the bladder, ovaries, and Fallopian tubes, in general, terminates favorably, especially when the pus has an outlet from the body, and the cause of the inflammation has ceased.

Gentlemen, in presenting to you these clinical considerations on perihysteric abscess, I have not intended to give you a complete description of all the inflammatory affections of the peritoneum and pelvic cellular tissue. In the actual state of science, I do not believe it possible to give such a description. Read the works on this subject of my much lamented hospital colleagues, the late Drs. Valleix,[1] Aran, and Goupil;[2] study with attention the memoir, so rich in facts, of Bernutz on pelvi-peritonitis, or orchitis in the female: you will then be convinced that the subject which I have been now discussing with you is one of the most obscure in pathology.

Be that as it may, I have pleasure in stating, that we are indebted to the authors I have now named for important views: it was a great contribution to science, to establish, that pelvi-peritonitis occurs frequently, and that perihysteric *phlegmon* is relatively rare.

I do not think it necessary to speak at any length on the pathological anatomy of pelvic abscess: the anatomical details given in my accounts of cases will suffice to impress on your minds the principal lesions.

Let me now describe to you a case which illustrates the multiplicity of pelvic lesions which may originate in inflammation of the uterus: and which also shows that the actual cautery, usually exempt from danger, may sometimes occasion mortal lesions.

A woman, aged twenty-seven, was admitted to Saint-Bernard's ward. She stated that she menstruated regularly, but always had leucorrhœa, and pains in the loins and lower part of the abdomen. By digital examination, it was found that the neck of the uterus was swollen, half open, and deeply excoriated. I resolved to apply the actual cautery. I generally employ it, when the lesions of the cervix are not superficial; and I have done so for more than fifteen years, without any bad results having been produced, till the case occurred which I am now going to relate. I applied the actual cautery to the cervix. Everything seemed to go on exceedingly well: the slough separated in a few days: the menses appeared in the interval, and four days after their cessation, I applied the actual cautery a second time, hoping that it would not be necessary to repeat the application. Five or six days after the second application, there was slight pain in the left iliac fossa, and deep-seated doughiness could be detected on palpation. Some days later, the pain increased, and there supervened neuralgia of the crural nerve, with contraction of the thigh on the pelvis. The iliac swelling became more apparent, the pains in the thigh increased, and the power of extending the limb ceased. On attempting extension of the limb, very acute pain was occasioned, shooting deep into the pelvis. Intense fever set in; and I saw clearly that there was phlegmon of the sheath of the psoas and iliacus muscles. A doughiness soon became appreciable below the crural arch, and pus burrowed to the trochanter minor. About five weeks after the last cauterisation, the unfortunate young woman died exhausted with fever and diarrhœa.

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[1] VALLEIX:—Guide de Médecin Practicien.

[2] GOUPIL:—Leçons Cliniques sur les Maladies de l'Utérus et de ses Annexes. Paris: 1860.

On examining the body after death, an abscess was found in the left broad ligament, slight peritonitis, and a purulent collection which had dissected the psoas and iliacus muscles, and extended from the lumbar region to the trochanter minor.

I have often asked myself, Gentlemen, whether the cauterisation occasioned the disaster; and I have answered the question in the affirmative. It is certain that if I had not performed that little operation, the iliac phlegmon had never been formed. But, Gentlemen, when I inquire into the results of my own practice, when I interrogate professional brethren who are not afraid to tell the truth as to their practice, I discover, that in a few rare instances, the most superficial cauterisation of the cervix has induced inflammation of the broad ligaments and peritoneum: I discover that not only is cauterisation with the red hot iron generally free from danger, but is even more generally harmless than cauterisation with potential caustics. As a general proposition, it is correct to say, that the deep sloughs produced by the actual cautery are a guarantee against serious phlegmons of the pelvis.

It must be admitted, that medicine is nearly impotent in the treatment of perihysteric abscesses. When the symptoms have informed us of the existence of phlegmon or peritonitis, I place exceedingly little reliance in the resolutive action of local or general bleeding. Generally, my only aim is to calm the pain by local applications, or the internal administration of preparations of belladonna and opium. By silencing the pain, it frequently happens that the inflammatory fluxion is moderated; and when I accomplish that object, I am well pleased with the result of the treatment.

Although we can do but little for the cure of pelvi-peritonitis and phlegmons of the pelvis, we can do a great deal in respect of the causes on which the inflammation depends. All our efforts, therefore, ought to be directed towards prevention, towards diminishing the influence of the causes, and studying the organic susceptibility before we interfere with chronic affections of the womb. For example, when menstruation is painful, and profuse menorrhagia testifies to the existence of an abnormal fluxion towards the genital organs, the woman ought to be advised to avoid everything likely to increase pain or hæmorrhage. Delivery, as I have said, is the most common cause of pelvic abscess; but we learn from experience, that unless an epidemic influence be prevailing, pelvi-peritonitis and perihysteric abscess are hardly ever observed, except in persons in whom labour has been abnormal or protracted, or in patients who have committed some imprudence within a few days of delivery. To state the etiological conditions, is to indicate the rules which ought to be followed to avoid the consequences of these conditions.

In the cases in which it is necessary to use caustics to modify inflammation of the cervix uteri, or in uterine catarrh, always proceed with extreme caution: always fear setting up an inflammation which may extend to the peritoneum. Interrogate—if I may use the expression—interrogate the susceptibility of the organ on which you are going to act; and before resorting to powerful caustics, use those which are feebler: stop your applications whenever you find they occasion acute pain.

Likewise, in specific inflammation of the mucous membrane of the vagina, abstain from interfering too brusquely by substitutive treatment. Recollect, that in these cases also, there is a risk of causing pelvi-peritonitis.

Gentlemen, in making a digital examination of the uterus, you cannot exercise too much caution. I have related to you a case in which a somewhat roughly performed digital examination occasioned pelvi-peritonitis, which lasted for three long years!

Never employ the hysterometer except in the exceptional cases in which catheterism is indispensable for diagnostic purposes. Such cases are of infinitely rare occurrence. In a still more emphatic manner, I denounce intra-uterine pessaries, which ought not to be employed under any circumstances: this opinion I the more hold to, from having been taught by experience, that deviations of the womb are almost never the cause of the pains and discomforts from which women suffer. These pains are generally symptomatic of metritis, uterine catarrh, or chronic pelvi-peritonitis, which are only aggravated by the introduction of a foreign body into the uterus.

When pelvic abscesses point in the vagina or rectum, they ought not to be opened. The physician ought likewise to refrain from active interference in cases of retro-uterine hæmatocele. On the other hand, when the perihysteric abscess has invaded the iliac fossa, operative interference is demanded. You are acquainted with all the grave complications of these abscesses when allowed to remain too long in contact with the psoas muscle and the cellular tissue of the iliac fossa: you know that they have a tendency to work their way towards the sacro-iliac and coxo-femoral articulations, and that when this takes place, the issue of the case is nearly always mortal. Consequently, as soon as you have become satisfied by an attentive examination, that there is pus in the iliac fossa, you ought to open the abscess by the bistoury; or at least, you ought, by the use of caustics, to hasten the formation of adhesions, so that you may safely make the opening. In these circumstances, you must not wait too long; for prolonged waiting may render ultimate interference completely useless.

LECTURE XCVIII.

NEW SPECIES OF ANASARCA, THE SEQUEL OF RETENTION OF URINE.

The Anasarca is observed, and the Retention of Urine is not recognised.—Relation of Cause and Effect between the Anasarca and the Retention is, with greater reason, not recognised.—The Distended Bladder may be mistaken for a Malignant Tumor.—Accumulation of Urine.—The Anasarca is rapidly cured by the Evacuation of the Urine.—Why Retention of Urine causes Anasarca.

Gentlemen:—I wish to avail myself of this opportunity of speaking to you regarding a species of general dropsy which accompanies retention of urine. Though we see less frequently in the medical than in the surgical wards patients suffering from affections of the urinary passages, yet it is not unusual for persons with anasarca to be directed to our wards, because dropsy is generally looked upon as the consequence of some grave internal lesion. Hospital surgeons themselves do not hesitate to send this class of patients into the medical wards, because they nearly all ignore the influence which retention of urine sometimes has in producing anasarca. For a similar reason, we physicians receive these patients in our consulting-rooms; whereas, had this species of anasarca been better known, they would have been sent to our colleagues who devote themselves to surgery.

Gentlemen, you have not forgotten the patient who, during the summer of 1864, was admitted to Saint-Agnes's ward, and there occupied bed No. 3. With me, you were struck by the great amount of anasarca in every part of the body. When, after examining the case for a minute, and discovering that the bladder was distended, I intimated that that condition was very probably the cause of the dropsy, which would disappear when the urine was drawn off by the catheter or passed freely, I observed a smile of incredulity on your faces. Gentlemen, the urine was not albuminous: the liver, heart, and lungs presented no abnormal indication: no trace of cancerous or tuberculous disease could be detected. Some days later, you were convinced that I was right: at all events, you admitted, that in that particular case I was not mistaken, and you learned from the patient who had not attached much importance to the circumstance, that for two months, micturition had been becoming more and more difficult; and that the patient had long had stricture of the urethra and disease of the prostate. When cured of his dropsy, I sent him into the wards of my colleague M. Maisonneuve, whose province it was to treat the affection of the urinary passages, the cause of the anasarca.

The diagnosis which I made with so much ease, and the favorable prognosis which I formed in this case, apparently so very serious, were, Gentlemen, neither the results of divination nor of chance. More than ten years previously, I had learned from Dr. Bourgeois (of Etampes) to recognise cases of this kind. In 1855, he described to me a very remarkable form of general dropsy, which he had observed after incomplete retention and insufficent emission of urine. I strongly urged him to send a note on the subject to the Academy of Medicine, which he did soon afterwards.

From that time, my attention has been keenly directed to the subject; and I have had frequent opportunities of verifying the lesson I learned from Dr. Bourgeois. Few years pass in which I do not see patients of this kind in the clinical wards, and still greater numbers of them come to me in my private consulting-room. I exceedingly regret that I have kept no note of these curious cases: but during the current year, within three months, I have seen four cases, one in my wards in the Hôtel-Dieu, one in my consulting-room, and the other two in the practice of two brother practitioners of Paris.

Before describing these cases which I have examined recently, allow me, Gentlemen, to read to you the account of the two cases which constitute the basis of the memoir which Dr. Bourgeois sent to the Academy of Medicine in 1855, which account he was so obliging as to communicate to me.

"About the year 1846," says Dr. Bourgeois, "I was called to the neighbourhood of Pithiviers, to a gentleman aged thirty, affected with dropsy, for which he had been under treatment by two of my brother-practitioners for some weeks. I found the patient in bed, to which I was told he was chiefly confined. The face was pale and swollen; but neither the features nor the colour of the skin were much altered. The whole body was greatly swollen: there was no cyanosis: the pulse was quick and compressible: the appetite was deficient: the thirst was great: inspiration became impeded in the horizontal decubitus. The abdomen, much distended, communicated the sensation of fluctuation; and by pressing on it somewhat strongly, a large, isolated, oval tumour was felt, which reached from above the umbilicus to deep down in the pelvis. The cellular tissue of the limbs and trunk were much distended. On examining the heart and lungs, no lesion was detected, except, that at the base of the chest, in the precordial region, there was dulness, due to the presence of a certain amount of fluid in the pleuræ and pericardium. I forgot to mention that the patient had never had a complete stoppage of urine: he was always passing urine unconsciously, and without any desire to urinate. His constitution was slightly lymphatic, but otherwise good. When in a heated state, he had bathed in very cold water, and had in consequence suffered from slight diarrhœa and colic in the lower part of the abdomen: then came the almost total inability to pass urine, which began at last to dribble from him constantly. Some time after this, seeing that his limbs were swollen, and being still more alarmed by the diminution of his strength, he summoned the doctor of Pithiviers: matters becoming worse, I was called in.

"The examination which I made of the abdomen led me to think that the cause of the anasarca was to be found in the state of the urinary passages; that the bladder was enormously distended; and that the patient had urinated by overflow from engorgement, which had misled my colleagues. In all such cases, as a preliminary measure, I propose the use of the catheter: in this case, the proposal was accepted by the patient and his medical attendants. Hardly had the silver catheter entered the bladder, than the urine gushed forth in a full stream, till at least three litres had passed. The relief was immediate: before we left the patient, the desire to urinate, absent for several weeks, was felt, but without the ability to satisfy it. The catheter was introduced a second time; and we were astonished at the quantity of urine evacuated: it was clear, limpid, and nearly without odour. A catheter was left in the bladder, which was unstopped, at the patient's request, every half hour. This system was continued for two or three days, during which period, the patient passed from twelve to fifteen litres of urine. Within the same period, the anasarca entirely disappeared: and all the functions were very speedily restored, excepting the voluntary emission of urine. The patient was obliged to draw off his water by the catheter, till his death; which occurred several years later, of cerebral disease.

"Some years later, I was sent for to Pussay, to visit a strong old man of sanguineous temperament, nearly seventy-five years of age, who for some months had had difficulty in completely emptying his bladder. This old man, urinating with increasing difficulty, and passing a certain quantity of urine unconsciously, seeing, moreover, that his limbs were swollen, called in a physician of Augerville, who considered the case as one in which there was incontinence of urine, and incipient dropsy, the latter affection not having any relation to the former. As matters increased in seriousness day by day, I was asked to meet in consultation the ordinary medical attendant. I found the patient in the following state: there was no sensible alteration of the features: there was infiltration of the face: the decubitus was dorsal: he was confined to bed: there was very little oppression in the breathing: the pulse was tolerably full, but compressible, and not quick: the appetite was impaired: the bowels were seldom opened: there was a constant dribbling of urine, by which great erythema of the surrounding skin had been caused: the state of general swelling was very marked.

"On applying some force in the palpation of the abdomen, there was discovered, in the hypogastric region, a large oval tumor, extending from the pubes to above the umbilicus: no desire to urinate was excited by pressure on the tumor. On seeing this poor old man, and hearing a short statement by his relations, I immediately recollected my previous case, and concluded, that the anasarca was due to insufficient micturition, and that there was also a reflux of fluid into the splanchnic cavities and interstitial tissue. I was convinced of the nature of the case by a most thorough examination, and immediately proposed catheterism: this was attempted in vain by a silver catheter, the only instrument at our disposal. After numerous trials, we were obliged to adjourn the operation till we were provided with more suitable instruments: but the family, one of whose member was at that time under the treatment of M. Ségalas, proposed that that able practitioner should be called in, a proposal to which we assented with satisfaction. Next morning, we were accompanied in our visit to the old gentleman by M. Ségalas. In vain, M. Ségalas attempted to surmount the obstacle by using metallic catheters and soft sounds of cylindrical, conical, and olivary shape: for a long time, he was unable to pass an instrument into the bladder, till, at last, he succeeded in introducing a curved catheter. The instrument had no sooner entered the bladder, than the urine issued forth in a rapid forcible stream. Without a pause, the patient passed several litres of urine. As the introduction of the catheter had been a matter of so much difficulty, we deemed it prudent to leave it in the bladder. Some time after the first evacuation—which was complete—the patient felt, for the first time, a desire to pass urine: the stopper was then taken out of the instrument, when, considering the short time which had elapsed since the bladder was emptied, a surprising quantity was discharged. Every half hour (or hour at the longest), the bladder was emptied by request of the patient; and, as in the previous case, the dropsy disappeared within two or three days. The power of urinating voluntarily never returned, although, with the exception of this inconvenience, he lived in completely restored health, till he died, four or five years afterwards, of a disease in no way connected with the affection now described. Till the end of his life, it was necessary to draw off his urine by the catheter. The operation was regularly performed by a member of the family, and always without difficulty. In this case, there was no real urethral obstacle—only a deviation at the prostatic portion of the canal."

I now proceed, Gentlemen, to describe the cases which have recently occurred in my own practice.

In July, 1864, a gentleman, aged sixty-four, consulted me in my cabinet. He ascended my stair with difficulty, and although he had rested more than an hour in the waiting-room, he was panting dreadfully when he entered my consulting-room. His face and hands were infiltrated; and forthwith, I discovered that the inferior extremities were equally swollen. The abdomen was very large.

The patient informed me, that the general swelling of his body had commenced two months previously, without any appreciable disturbance of his health except severe abdominal pains. The anasarca began in the legs; and in eight or ten days, had extended to the whole body. The physician in attendance had then ascertained that there was a tumor in the abdomen; and in sending his client to me, he directed my attention to this tumor, which he regarded as the starting point of all the symptoms.

Ere long, the swelling increased enormously; and at last the patient's miseries culminated in orthopnœa.

I conjectured that he had Bright's disease; and asked him to give me some of his urine. He passed urine in my presence, without much difficulty; but the quantity passed was small. I found neither albumen nor glucose in the urine. I auscultated the heart and lungs, without finding anything to explain his serious condition.

Having caused the patient to lie down on the sofa, I very carefully examined his abdomen. I there found an enormous tumor, reaching from the pelvis to above the umbilicus. It was elastic, and perfectly round. I at once perceived that it was the bladder. Without saying anything to the patient, I introduced a catheter into his bladder, which I did without any difficulty; and withdrew three litres of limpid urine. The tumor forthwith disappeared.

I then inquired into all the circumstances prior to the invasion of the symptoms which had led him to consult me. I learned facts which had not been stated to me in the first instance, as the patient's attention had not been directed to them.

For two or three years, his bladder had been sluggish. He urinated frequently, both by day and by night; and on every occasion, he had to use efforts. A month before the commencement of the general dropsy, he observed that he could not urinate when in bed, lying down: he required to kneel. A few days later, he became obliged to get out of bed to make water. After some days passed in this state, he became unable to urinate on first taking up the vessel: he was obliged to walk, barefooted, up and down his room for some minutes ere any urine passed; and then, it was only with great straining, that he could pass above half a tumbler.

It was at this stage, that his feet began to swell. He had then called in the physician who had found in the hypogastrium a very hard tumor which he considered a malignant growth. He had inquired about the urine, and had been told by the patient that his urine was rather more abundant than when he was in a normal condition: the physician, consequently, paid no more attention to that matter. Strange to say, when the patient came to me, he did not mention his urinary difficulties; and it was not till after I had used the catheter, that I obtained from him the details I have now given you—in fact, not till after I had, to a certain extent, guided his recollections.

I saw clearly, that all the dropsical symptoms were consequent upon the retention of urine. As he had an affection of the prostate, and as I felt that I was quite incompetent to deal with such an affair, I placed him under the care of a colleague, more able than me, who taught him to introduce the catheter. As soon as the evacuation of the urine was regularly attended to, the anasarca disappeared, as well as the dyspnœa and other serious symptoms under which the patient had suffered for two months.

On the 5th September, 1864, I saw, in consultation with Dr. Lepère, a case of which I am now about to give you a very succinct account.

The patient, a man of fifty-five years of age, was very subject to hæmorrhoidal flux, by which he had been greatly weakened. From time to time, during the three preceding years, he had experienced, during the night, a stoppage in the stream of urine of which he did not take much notice, attributing it to spasms induced by the hæmorrhoids. During the summer of 1864, he often felt difficulty in passing his urine: there was an obvious change in his state of health: he became thin, and his appetite was impaired. In the mean time, during July, he went to Ems with his wife, for whom the waters had been prescribed.

About the 15th of August, M. Lepère was informed by letter that the patient had suffered dreadfully from his hæmorrhoids; that hands, legs, and abdomen were swollen; and that the physicians had discovered a cancerous tumor connected with the liver. Our Parisian colleague replied, that the latter statement could not be correct, for he had, a month previously, very carefully examined the abdomen by palpation, and had then ascertained that the organs were in a normal state. In answer, M. Lepère received an assertion still more categorical than the first. When the wife had concluded her course of the waters, the husband was sent to Kissingen. There, the severity of the symptoms increased rather than diminished. M. Lepère received a letter, in which he was requested to invite me to meet him in consultation in the case on the 5th September.

We found the patient seated. He was suffering considerably from oppressed breathing: the face was pale and puffy: the hands were œdematous: the legs and abdomen were enormously swollen.

Before making a more minute investigation, we asked for some urine: it was clear, and did not give an albuminous precipitate when treated by heat. When we proceeded to palpation, we at once found that there was a large, round, elastic tumor in the hypogastrium and reaching far above the umbilicus: in size and form, it resembled the uterine tumor at the eighth month of pregnancy. The tumor, I perceived, was evidently the bladder distended with urine; and I had no difficulty in seeing that the case was similar to those reported by Dr. Bourgeois, and to others like them which I had myself observed. A catheter was introduced into the bladder, when eight litres of urine were withdrawn.

It is unnecessary for me to add, that the tumor entirely disappeared. After four days of the use of the catheter, the swelling was gone; and the secretion of urine was very abundant.

It becomes very interesting to inquire how it was, that this patient, a man of great intelligence, and very observant of his different symptoms, should have forgotten all that pertained to the bladder in the history of his disease. When we had brought back his recollections to their proper bearings, the patient told us that since his sojourn at Ems, he had been obliged to rise every night, and even to walk about the room when he wished to urinate: he had never had retention of urine, and the night preceding my visit, he had urinated voluntarily five or six times, each time passing one hundred and fifty grammes of urine.

Gentlemen, I dwell on these circumstances, because this patient, like the other whose case I have described to you, never spoke of retention of urine—never even mentioned having suffered from any inconvenience referable to the urinary passages. It was only by palpation of the abdomen, that a clue to the diagnosis was obtained.

My part in the case of M. Lepère's patient was at an end. As he had an affection of the bladder, M. Ricord was called in. He found that there was a very large calculus; but did not consider it advisable to attempt lithotrity. In two months, the patient died, with symptoms of purulent infection depending on extensive disease of the prostate.

During the same month, I saw a patient living in one of the streets of the Marais, to whom I was called by one of my colleagues. There was general anasarca, which had lasted nearly six weeks. The patient was a man about sixty years of age, who had had sluggishness in urinating, but no incontinence of urine. My colleague who had perfectly ascertained that there was repletion of the bladder, feared, however, from the existence of dropsy, that there was serious organic disease. I related to him some of the cases which I had seen; and I had no difficulty in getting him to adopt some of my ideas as to the absence of danger. It was agreed that the catheter should be used several times a day. Soon afterwards, I learned that the anasarca had disappeared from the time that free passage had been afforded to the urine.

Recently, when in company with Dr. Follin, I was speaking to him of the singular relation between retention of urine and general dropsy, when he mentioned to me that he had been called to the country to see a patient whom he found in the conditions similar to those I have been describing; and that he had been surprised and alarmed at the anasarca which existed.

It is quite certain, then, Gentlemen, as stated by Dr. Bourgeois, that retention of urine may occasion general dropsy. The recognition of this fact has a very important bearing on practice. We know that anasarca may show itself in the form in which it usually appears in Bright's disease, and yet be as easy of cure, as it is inexorably rebellious to treatment when associated with that malady.

Again, there is the curious fact, that many patients are not aware that they have retention of urine. When we see dropsy supervene slowly, extending even to the face, and when, at the same time, the health of the patient becomes deteriorated in a manner similar to that which takes place in diseases of the urinary organs, even in those which arise unknown to the patient, it is very difficult not to believe in the existence of some serious organic mischief; and even after it has been ascertained by abdominal palpation, that the bladder is distended by urine, one would be apt to attribute the retention to a malignant tumor, were it not for the favorable view suggested by such cases as I have just laid before you.

Gentlemen, it is difficult to explain the manner in which the species of anasarca now under consideration is produced. I have often asked myself whether, when the urine, this excrementitious fluid, has been long pent up in the bladder, it does not flow backwards by the ureters into the pelves and calices of the kidneys, so as to distend the organs and impede their function. By the blood being thus prevented from throwing off its excess of water, which flows abundantly to the renal surface, general dropsy is produced. I have a great dislike to this mechanical explanation, and hazard it with much timidity. Perhaps we are justified in supposing, that the compression of the kidneys prevents that complete emunction which they ought to perform; and that, in consequence, the constitution of the blood undergoes a great alteration—an alteration which ceases on removal of the cause.

For the present, Gentlemen, let it suffice, that you know, that there is a species of anasarca due to retention of urine; and that you learn to detect it, and treat it.

LECTURE XCIX.

MOVEABLE KIDNEY.

Frequency of Moveable Kidney.—Reason of this Frequency is the Feebleness of the Attachment of the Kidneys.—Frequency greater in Women than in Men; and on the Right than on the Left Side.—Explanation.—Moveable Kidneys are not always Painful.—How they become Painful.—Numerous Errors of Diagnosis: Means of avoiding them.—Treatment.

Gentlemen:—You have seen very recently among the out-patients at the Hôtel-Dieu, a man thirty-five years of age, robust in appearance, of powerful muscle, and presenting every condition of good health, who complained, nevertheless, of having an abdominal tumor, and of suffering from *peritonitis*, to which affection he said he was very subject. A tumor in the abdomen is very unusual, and peritonitis is still more unusual, in a man who generally enjoys good health. The man's face expressed pain, but had not a pinched appearance; and he had no fever. The possibility of displacement of the kidney at once presented itself to my mind; and I told the patient to take off his clothes. You saw that the abdomen was furrowed by the cicatrices of cuppings and leechings, showing that the patient had many times previously suffered from similar pains, and had on each occasion been treated for peritonitis.

Although thickness of the abdominal parietes rendered exploration difficult, and although it was rendered still more difficult by the pain, I could easily recognise, and enable you to verify for yourselves, the existence of a tumor in the right side. It was hard, shaped like an orbicular ring, and rather painful. It could be easily moved backwards and forwards, but it could not be brought to the median line. By steady gentle pressure, it could be pushed into the right renal region.

The patient was free from fever, and consequently free from inflammation: pressure on the abdomen did not occasion pain, consequently, there was no peritonitis: there was neither vomiting nor difficulty in passing water, consequently, neither nephritic colic nor renal lesion existed. The tumor in question, moreover, had really the form of the kidney, and could be pushed into the renal region. In your presence, I made a little experiment, which was quite a demonstration. Pressing on the tumor, I caused a certain amount of pain: placing then the hand on the left renal region, and making similar pressure, I produced pain in all respects similar, the patient said, to the pain occasioned by pressing the tumor in the right side. The tumor, therefore, could be nothing else than the right kidney: in fact, the case was one of moveable kidney, and not of peritonitis as had till then been supposed.

I ordered this man to wear a concave cushioned bandage, for the double purpose of supporting the kidney and protecting it from being injured by external objects. I also advised him to abstain from all antiphlogistic treatment. I recommended use of baths and poultices when the kidney was painful.

You will remark, that in this case it was the right kidney which was moveable. It is that kidney which is generally affected by mobility and displacement. You will also remark, that the patient was a man: according to statistics, the subjects are more frequently women.

Why do the kidneys become moveable? Why does the right become more frequently moveable than the left kidney? Why does the moveable kidney so easily become the seat of pain? These are questions which I wish to discuss with you.

Gentlemen, we cannot too carefully study the marvellous arrangements which nature has contrived for the protection of our organs. There is a simplicity of means, and a grandeur of results, presenting an aggregate which I am never weary of admiring. Arrived though I be at that period of life when the susceptibility to enthusiasm is pretty well past, I still feel an enthusiastic admiration for the works of nature.

Protection of organs is one of the fundamental principles which preside over the structure of the living body. Every organ requires to be protected from the objects in the external world; and yet, every organ requires to communicate with the external world.

The brain dwells and moves within a box of bone which is both thin and strong. It is thin, that its weight may not impede the movements of the head: it is strong, in virtue of its spheroidal form; and all the bones of which it is composed are dovetailed into one another by a series of indentations which diminishes strain, and deadens shock. The spinal cord is likewise protected by a bony case, the vertebral canal, which, with very great strength, unites elasticity to give power of resistance, and flexibility to impart ease of movement. You know to how great an extent the lungs are capable of expansion within their cage formed by flexible resisting arcs, the ribs, and by elastic planes, the intercostal muscles. Within the same cage, the heart is contained. The liver lies on the right side, hidden behind the lower false ribs, and under the diaphragmatic arch. The spleen lies on the left side, protected by the lower false ribs. The kidneys rest on thick muscular masses, formed by the quadrati lumborum and the origins of the psoas muscles. On the inside, they are protected by the vertebral column; and on the outside and posteriorly, by the quadrati lumborum, transverse processes of the lumbar vertebræ, the sacro-lumbar muscular mass, the spinalis dorsi, and very strong aponeuroses: in front, the intestinal convolutions separate them from the abdominal parietes. The bladder and uterus are protected inferiorly and posteriorly by the pubic girdle, within the cavity of the pelvis. The intestines alone seem to be badly protected: they have only the protection of a muscular wall. But then, as they are subject, by the requirements of digestion, to alternate expansion and contraction, it is essential that they be placed within a cavity which is extensible like themselves. Here, nevertheless, protection is also assured: to permit the free transit of their contents, the intestines are distended with gas: these gases form an elastic, and consequently a protective cushion. To promote the passage from above downwards of the contents of the digestive canal, that canal is endowed with an incredible facility of movement; and to this same facility of movement, it owes its protection from injury by shocks. You are acquainted with the great instinctive contractile power with which the abdominal muscles are endowed. When palpation of the abdomen is performed without due precaution, there is immediately perceived a stiffening, caused by an automatic contraction of the muscles, which comes to the protection of the subjacent organs. Within the abdomen, it appears then, that we find only those organs which are soft and elastic, the stomach and intestines, in relation with the soft abdominal parietes: all the hard organs—the liver, spleen, kidneys, and uterus—are placed deep, where they are protected by osseous bulwarks. From their solidity, pressure on them occasions pain and injurious consequences. You now perceive why it is, that the kidneys when moveable are exposed to pressure and painful friction, giving rise to many untoward symptoms.

Are moveable kidneys always the seat of pain? To be able to answer this question, it would be necessary to examine the kidneys of all the subjects submitted to our medical investigation. This has been done by Dr. Walther, an accomplished physician of Dresden; and I will tell you forthwith the results of his researches. I hardly ever diagnose moveable kidneys. When I do diagnose them, is when an individual suffering from them comes to me complaining of them. As he complains to me of symptoms caused by moveable kidney, I conclude that a moveable kidney is a distressing or painful affair. This, however, is a very illogical mode of reasoning; and yet it is one too frequently employed in medicine. It is by reasoning in this way, that the fatal mistake has arisen of supposing that deviations of the uterus are the cause of the pain, the pain being in reality due to the concomitant metritis. A woman complains to a physician of pain in the uterus, and manifold discomforts: he makes a digital examination, ascertains that there is a particular deviation of the uterus; and, ignoring the metritis, he concludes that the deviation is the cause of the uterine symptoms. But investigate by another mode. Follow the method of inquiry pursued by M. Gosselin at the Hôpital de Lourcine: he submitted indiscriminately all the women at that institution to digital examination of the uterus, those who were and those who were not suffering from a uterine affection, with the result of ascertaining, that every kind of deviation of the uterus was of very frequent occurrence: he found, moreover, that deviations of the uterus exist in women who do not suffer the very slightest uterine discomfort. Consequently, when women who suffer from the uterus have a deviation of the organ, their sufferings must not be attributed to that deviation, but to some other uterine affection, the deviation being in itself a very harmless affair.

Were all physicians accustomed to scientific precision, they would feel, that it was not logical to arrive at a conclusion until they had examined all data, and instituted experiments as counter-proofs. In the question now before us, the counter-proof consists in the examination of the kidneys of a great many persons not suffering pain. This, Dr. Walther has done: and he has obtained the curious result, that the kidneys are moveable in a considerable number of persons who suffer in no degree whatever therefrom, who give no thought to the peculiarity, and are even ignorant that they have a moveable kidney.

Moveable kidneys generally become painful as a consequence of great and unusual pressure, of a blow, or of prolonged fatigue: and it is after such occurrences that the patients come to us with their complaints.

My *chef de clinique*, Dr. Peter, was one day summoned to a client, a robust man, usually in the enjoyment of good health. He was a distinguished architect, a person of great intelligence, who gave a very good account of his sensations. He stated, that from the evening of the preceding day, he had been suffering acute pain in the right side of the abdomen, particularly at one special place which he indicated. Dr. Peter, placing his hand on that place—being well instructed in the affection by a recent visit to Dresden—had no difficulty in recognising, that at the special seat of pain there existed a tumor, that the tumor was moveable, and that the moveable tumor was the right kidney. But how was it, that this kidney, assuredly moveable for a long period and not till then painful, should have suddenly become the seat of pain? Dr. Peter inquired whether the patient had ever received a blow on the region, or had worn too tight a garment. At once enlightened by the questions, the patient stated that, on the previous evening, he had been on duty as a national guard, which duty he had, he said, unfortunately to perform about once in six months; and still more unfortunately, since his last time of service, he had become much fatter, so that he had had great difficulty in putting on his military trowsers, now too tight for him. He persisted however: the result was discomfort, which went on increasing till the following day, when it had become positive pain. Excuse the apparent triviality of these details: they teach their own lesson.

The friends by whom the patient was surrounded were already speaking of the application of leeches, because on a previous occasion they had been employed for similar symptoms. Dr. Peter, however, only prescribed the application of a poultice over the very sensitive kidney, a protracted general bath, and rest in bed for twenty-four hours. As he had foreseen, all the symptoms then ceased. As the patient was robust and stout—as his abdominal walls formed a thick cushion for the displaced kidney, and as generally, he suffered neither discomfort nor pain, Dr. Peter did not order a bandage to be worn, reserving that measure to be resorted to should any unpleasant symptoms supervene.

A very distinguished physician, Dr. Becquet, has published opinions which I believe to be correct on the pathogeny of moveable kidneys, and on the causes which occasionally render painful those moveable kidneys which are generally free from pain. He thinks that during the catamenial fluxion, the kidneys participate in the congestion of the genital organs, and become tumefied. This would explain the pains in the renal regions so frequently felt at the menstrual periods, particularly by women who are not regular in their courses. Being thus swollen and heavy, the kidney, particularly the right kidney, acquires a tendency to surmount the feeble obstacles by which it is retained in its place. When the congestion ceases, the organ returns to its original position; but similar congestion renewed again and again, pushes it farther and farther each time from its place. The kidney, at each congestion, becomes permanently heavier, and so descends into a lower position. In this way, says Dr. Becquet, the kidney, by slow degrees, but not without causing suffering, at last appears free and floating in the abdomen.[1]

In support of this statement Dr. Becquet mentions the case of a woman in whom, at each menstrual period, the kidney, swollen and very painful on pressure, could be felt bulging under the ribs. As soon as the periodical congestion ceased, the kidney regained its usual volume, indolence, and place. The moveable character became permanent at a later date; and (as formerly) at each menstrual period, the now moveable kidney became the seat of pain. On one occasion, the renal fluxion being excessive, partial peritonitis arose, followed by the formation of false membranes. This resulted in the displaced kidney ceasing to be moveable, and becoming definitively fixed in an abnormal position.

My accomplished friend and colleague, Dr. Gueneau de Mussy in the excellent lectures which he gave on this subject at the Hôtel Dieu, quite adopted Dr. Becquet's opinion, and stated that he had met with a case which supported it. Dr. Gueneau de Mussy, however, added, that while he quite recognised that congestion may supervene, sometimes as a pathological condition, and sometimes as an epiphenomenon in the malady we are now considering, it must be admitted to be neither a constant cause nor a necessary complication, because floating kidneys are not uncommon in men, in whom their displacement cannot be attributed to any fluxionary process.[1] The correct view of the question could not be better expressed.

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[1] BECQUET:—Essai sur la Pathologie des Reins Flottants. [*Archives Générales de Médecine. T. I,* 1865.]

Dr. Gueneau de Mussy almost believes that hysteria and arthritis, though not causes of displacement of the kidneys, are causes of pain in displaced kidneys. He states, that he has met with moveable kidneys most frequently in hysterical and gouty persons.

My pupil, Dr. Peter, has just met with a case, similar to that reported by Dr. Becquet:—A robust negress, nurse in a family belonging to the Havanah, on returning fatigued, one day from the Exposition Universelle, was seized with pains in the right side. This occurred on the last day of her courses, which had been as abundant as usual. The pain became very acute; and the patient when she walked, was doubled up in two. The family was alarmed: Dr. Peter was sent for. When he arrived, the pains had continued for about twenty-four hours. He ascertained that there was an oval tumor in the right renal region, directed obliquely towards the hypogastrium, passing the umbilicus, and reaching within three finger breadths of the pubes. This last mentioned condition excluded the idea of pelvic hæmatocele, even though digital examination had not demonstrated the absolute integrity of the entire circum-uterine region. The tumor was hard, painful on pressure, almost immoveable: it was about twenty centimeters long and about ten centimeters in breadth. Dr. Peter had no hesitation in concluding that the tumor was a kidney out of place, bulky, congested during menstruation, and become painful, probably in consequence of fatigue from too much walking. The pain increased for three days; and there supervened, as in Dr. Becquet's case, slight circumscribed peritonitis. It was necessary to apply leeches twice, and to employ often repeated baths for fifteen days. The pain then yielded. The size of the tumor diminished a little: its limits could be made out very exactly by palpation, without giving the patient any pain. The examination *post morbum* confirmed the accuracy of the diagnosis. It was unquestionably the right kidney which had been painful and swollen. It remained in its abnormal position, out of which it could not be moved up more than two centimeters, and could not be restored to its proper anatomical position, which remained unoccupied. The pain had quite left the patient; and for some days after ceasing to be confined to bed, she made no complaint except of a feeling of weight. There was no return of pain at the next monthly period. Subsequently, Dr. Peter made several examinations, and satisfied himself that the displacement of the kidney continued. It is well to add, that there was nothing abnormal in the state of the urine during the period of the pain: it may have been a little less abundant than natural; but it never contained albumen, blood, nor pus.

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[1] Gueneau de Mussy:—*Union Médicale*, 1867.

I very recently saw, in consultation, a patient suffering from displacement of the right kidney. The nature of the affection had been mistaken by his ordinary medical attendant, who was one of my colleagues in the service of the hospitals, and an exceedingly well informed man. He thought that there was an abdominal tumor, which was true; and he used every effort to dissolve it by iodide of potassium without succeeding, which was fortunate. Notwithstanding all the treatment, or rather in consequence of it, the tumor became very painful, and gave great annoyance to the patient, who suffered also from the conviction that he had an abdominal tumour which could not be dissolved, and was destined to have an evil issue. The poor man had in fact fallen into a most dismal state of hypochondria. I had not much difficulty in satisfying the physician as to the real nature of the tumour, nor in satisfying the patient that his case was not one of much gravity. The only treatment required was the use of a suitable bandage.

You observe, that six of the patients of whom I have spoken to you were men. Dr. Rayner, however, has observed that displacement of the kidney is much more frequent in women than in men: and of thirty-five cases collected by Dr. Fritz, thirty occurred in women.[1]

An attempt has been made to explain this greater frequency of displacement of the kidney in women, by their use, or rather their abuse, of the corset. That is the opinion of my accomplished colleague Professor Cruveilhier. "Displacement of the kidney," he says, "occurs when pressure by the corset is made on the liver: the kidney is then driven out from the sort of recess which it occupies in the anterior surface of the liver, very much as a nut is, by pressure, squeezed out from between the fingers." Professor Cruveilhier adds:—"The left kidney is less frequently displaced than the right, because the left hypochondrium, occupied by the spleen and great curvature of the stomach, supports much better than the left hypochondrium, the pressure of the corset."

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[1] Fritz:—Des Reins Flottants. [*Archives Générales de Médecine.*] 1859. p. 158.

In the case of men, this explanation is not admissible. Moreover, I am far from believing that a change in the volume of the liver can be a frequent determining cause of pressing down, and of consequently producing mobility of, the right kidney.

Let me lay before you the account of another case of displacement of the kidney in a woman, for which I am indebted to Dr. Peter.

A woman, about thirty years of age presented herself in the out-patients' consulting-room at the Hôtel Dieu. She was tall, well made, and presented all the characteristics of the nervo-lymphatic temperament. The face, however, had an almost waxy paleness: the sclerotics were bluish: and the general expression of the countenance indicated long continued suffering.

The first thing of which this woman complained was a *tumor of the liver*, a tumor the existence of which had been long previously ascertained by many physicians, who all concurred in regarding it as incurable. She had never had the paroxysm of pain which characterises hepatic colic, nor the jaundice which is its sequel. She had never suffered from dyspeptic symptoms, invariably associated with organic disease of the liver. She had never had epistaxis. Her general state did not in any way indicate that radical alteration of the organism always observable when a grave lesion of the liver has existed for three years.

Dr. Peter, deferring for greater leisure his examination of the liver, pursued his inquiry with a view to elicit, whether there existed any other cause to explain the anæmic cachexia indicated by the waxy paleness of the face. He soon discovered that the patient had been confined three years previously, that after delivery, she had had metritis, that that metritis became chronic, and caused profuse menorrhagia at each menstrual epoch. Having ascertained these facts, Dr. Peter placed the patient on a bed, and methodically examined the abdomen by palpation. He found that the liver extended three finger breadths beyond the false ribs in the mammary region, that its vertical diameter at that point was twelve centimeters, which is nearly normal. A little lower down, there was felt a hard ovoid tumor, the upper margin of which was in juxta-position with the liver. This was the only relation which the tumor had with the liver: there was no point at which it adhered to the liver. The tumor could be made to float in the abdomen, so as to be brought to the umbilicus: it was very painful to the touch. Dr. Peter had already formed his opinion; but to have superabundance of proof, he pressed his hand down into the region of the right kidney, and found it empty. This settled the question beyond any possibility of doubt. To make the matter more evident to pupils, he delineated both the liver and the moveable tumor with his plessigraph: it could then easily be *seen* that the liver had its normal form, that the tumor had the ovoid form of the kidney, and that there was a space of nearly two centimeters between the most accessible margin of the tumor and the lower surface of the liver, from which a tympanitic sound was elicited on percussion. It was, therefore, evident that the tumor in question did not adhere to the liver, and was in fact the displaced right kidney.

Consequent upon delivery, the abdomen of this woman had become very flaccid; and it was some time after her confinement that she experienced, for the first time, pretty severe pains in the right side. These pains were not accompanied by vomiting, nor by any general disorder of the system. Some time previously, this woman had become a servant in one of Duval's eating-houses. As you know, the female servants in these establishments are constantly on their legs, running from table to table, and quickly going up and down stairs. Cannot you now understand how the right kidney, already moveable perhaps, had become definitively displaced from not being adequately supported by the relaxed abdominal parietes?—how this displacement had increased by an occupation in which the displaced organ was being constantly subjected to shocks in the movements of the body?—and how the pains should be more severe than usual after a day of more than usual fatigue? It is well to remark that the patient simultaneously gave all these details—details which, when rationally grouped, throw a flood of light on the etiology of the affection.

The most curious circumstance in relation to the diagnosis of this case, is that the woman—by her own account—had been examined by more than ten physicians, and that all, with one exception, were of opinion, that she had a malignant tumor of the liver. The physician who dissented in opinion from the others had a still more strange view of the case. Without taking into account the seat of the tumor, its form, and the circumstance that its distance from the pubes was more than ten centimeters, he believed it to be a tumor of the uterus. True, this doctor was a homœopath. He treated the metritis, which really existed; but he cured neither it nor the tumor. The unfortunate woman had to pay him two hundred francs.

Being a very intelligent person, she had observed, that when her abdomen was sufficiently supported, she either suffered very little, or not at all, in the tumor, from the fatigues of the day. Acting on this observation, she had a belt made for herself, which was rather ingeniously constructed, but too tight. Dr. Peter had no difficulty in getting her to make another in place of it, of strong drill, formed so as to embrace all the soft parietes of the abdomen, and capable of being laced and unlaced at pleasure. This girdle, in the situation corresponding to the tumor, was provided with a concave elastic cushion, intended at once to support and fix the tumor.

When we think of the feeble means by which nature has fixed the kidneys, it is rather matter of astonishment that they are generally retained in place, than that they are sometimes displaced. They are appended to the vascular system by the renal artery and vein—a feeble means of attachment, it must be admitted. The circum-renal cellulo-adipose tissue cannot keep the kidney fixed in its place. In reality, there is only the peritoneum to keep the kidney in apposition with the quadratus lumborum and psoas muscles; and this statement is proved to be correct by the facility with which the organ is removed when the peritoneum is torn. But the peritoneum is an agent very inadequate to fix the kidney. You perceive, then, that a sufficient reason for displacement of the kidney exists in the feebleness of the attachments which retain it in its position.

It is quite evident, that with such anatomical predispositions to displacement, increased volume, necessarily involving increased weight of the kidney, must almost inevitably lead to its position being lowered, and its being rendered moveable. Hydro-nephrosis is also a cause of displacement of the kidney: cases of this kind are mentioned by authors. This affection did not exist in any of my patients; and the great relative frequency of moveable kidney ascertained by Dr. Walther can only be explained by the feeble fixings of the organ.

As to the *symptoms* of this ectopia, the truth is, that in the great majority of cases, there are none: that is the conclusion derived from Dr. Walther's researches. Sometimes, the individual affected accidentally perceives a hard moveable tumor, which is painful on pressure. In such a case, when the physician is consulted, he will be able to ascertain that the tumor is a smooth and ovoid body, presenting the physical characters of the kidney. In most cases, the entire outline of the organ cannot be traced out: generally, we can only feel the upper part, from the organ lying in a direction obliquely from behind forwards, and from without inwards. This tumor is dull on percussion. By skilful palpation, it is discoverable that the renal region, on the side of the tumor, does not contain a kidney. The examination will be specially convincing, if we explore in succession the two renal regions. Such an investigation serves as a counter-proof confirmatory of the diagnosis.

Functional disturbance is absent in the majority of cases: when present, it consists in general discomfort, a feeling of weight, dragging, and pinching, but rarely amounting to actual pain. When pain does exist, it is generally of a dull character: sometimes, it causes depression of spirits. At times, patients speak of feeling as if one of their organs was detached [*décroché*], and floating free in the abdomen.

Whatever painful sensations are experienced by the patients, it is evident that they must be aggravated by great muscular exertion, prolonged or rapid walking, dancing, riding, and jolting in an ill-hung carriage. It sometimes happens, that the first uncomfortable sensations are experienced under the influence of one or other of these causes. At other times, as in the second case which I described to you, pain was first set up by the pressure of a too tight garment.

As direct or indirect symptoms of moveable kidney, Dr. Gueneau de Mussy likewise enumerates lumbar hyperæsthesia, pleuralgia, and dyspeptic symptoms. That eminent clinician remarks, that rest, and the horizontal decubitus, subdue the symptoms which have been augmented or renewed by walking, jolting in a carriage, or the disturbance excited in the system by menstruation or the gouty fluxion. At other times, the pains manifest themselves in remote parts—in the thigh, epigastrium, or lower intercostal spaces, for example. Dr. Gueneau de Mussy considers these pains as reflex phenomena, the starting point being the kidney, and which, through the ganglia of the sympathetic and spinal cord, are transmitted to the spinal nerves.

Dr. Gueneau de Mussy believes that hypochondria, particularly in gouty and hysterical persons, is a possible consequence of pains depending on reflex action in cases of moveable kidney.

Neither the secretion of urine nor micturition are in the slightest degree influenced by the kidney being moveable.

As nothing frightens patients so much as the existence of an abdominal tumor, particularly when the physician appears ignorant of the true nature of the tumor, and when his endeavours to disperse or diminish it are too clearly unavailing, ectropia in a certain proportion of cases, induces melancholia and hypochondriasis. Such was the state of my patient, when I saw her in consultation.

Moveable kidneys may cause endless errors in diagnosis. Our Hôtel-Dieu out-patient, you have seen, was supposed to have had a series of attacks of peritonitis. In the private patient whom I saw with my colleague, it was supposed that there was a malignant tumor.

Professor Cruveilhier says:—"I have seen the tumor formed by the displaced right kidney treated as an *obstruction of the liver* or a *morbid* growth." Dr. Rayer says:—"The pains which sometimes accompany mobility of the kidney have been mistaken for *nervous colic*, for the phenomena of *hypochondriasis*, and sometimes even for *lumbar and sciatic neuralgia*." You will recollect that my private patient had become hypochondriacal.

Let me now explain to you the manner of searching for a moveable kidney. The physician placing himself at the side of the ectropia—the right side let us suppose—will glide the left hand along the margin of the lower false ribs, between them and the crest of the ilium: with the right hand, he will then slowly depress the wall of the abdomen, and so push aside the intestines and be thus enabled to reach the displaced kidney, and get it between his hands. In this way, he will be able to ascertain what the organ is, and also its abnormal mobility. By following the mode of examination which I have now described, Dr. Walther has ascertained that mobility of the kidney is exceedingly frequent; that it is a condition which generally remains unknown to the individual affected by it; and that it is a source of innumerable mistakes.

The tumor constituted by the displaced kidney may be mistaken for a tumor of the liver, gall-bladder, spleen, mesentery, intestine, or for a fibrous tumor of the ovary. The intestinal tumors for which it may be mistaken are invaginations, and collections of fæces. Lumbar pains arising from displaced kidney have often been supposed to depend on metritis in women who had leucorrhœa along with the renal ectropia.

It will, however, be sufficient to search the renal region on the side on which the tumor is situated, to determine, whether or not it be empty; and to find out, whether (as in the Hôtel-Dieu patient) pressure on the tumour and on the kidney, which is *in situ*, does not produce exactly similar sensations. A tumor of the liver is not moveable. When the spleen is low down in the abdomen, it is more voluminous than the displaced kidney. Intestinal tumors give rise to symptoms which are special and characteristic. A fibrous tumor of the ovary is indolent. Digital examination, if required, and the use of the speculum, by revealing the state of the uterus, will enable us to set down to their true cause the pains attributed to metritis.

In itself, displaced kidney does not present any gravity in the prognosis. The prognosis only becomes serious through blunders in diagnosis, and consequent improper treatment—treatment generally the more active, the less sure the physician is as to the nature of the affection. You have seen the abdomen of our Hôtel-Dieu patient covered by numerous cicatrices of leechings and cuppings, resorted to for the treatment of imaginary peritonitis. Taking blood from that man was useless: it weakened him, and did not cure him. It was equally useless to employ blisters and resolving ointments in the case of the private patient whom I saw in town. The too evident want of success of these measures in that case was shown by the confusion of the physician, and the hypochondriasis of the patient.

When the existence of renal ectropia has been discovered, its treatment follows as a necessary deduction. A prior indication must, however, be fulfilled—the kidney must be replaced. But to do this is almost impossible. In the case of women, however, who lace the corset too tightly, there is reason to hope that the kidney will return to a certain extent to its normal situation, upon a system of more moderate constriction being adopted. There are two secondary indications which remain to be stated:—to support the kidney, and to protect it. One apparatus will fulfil both intentions. The patient may be recommended to wear either a broad belt formed of woven elastic caoutchouc material, similar to that of which stockings are made for persons having varicose veins; or they may be advised to wear a belt made on the plan of the hypogastric belt, furnished with a slightly concave cushion adapted to the particular case. I found an apparatus of this description succeed in the two cases of which I have been speaking to you. I need not say that the bandage must be provided with thigh straps, to prevent it rising up out of its place. It is unnecessary to say more on this means of support. When you have made your diagnosis, you will easily find the means of supporting and protecting the displaced kidney when it is the cause of pain. I have said nothing of rest, baths, or cataplasms: but quite understand, that they must be resorted to when required. What I particularly wish you to bear in mind, is the frequency of ectropia of the kidney; and the frequency with which it leads to mistakes not less injurious to the reputation of the physician, than to the health of the patient. Finally, moveable kidney is an infirmity which is not serious, which we can always hope to alleviate, but hardly can ever hope to cure.

LECTURE C.

LOOSENING OF THE PELVIC SYMPHYSES.

Condition which is generally mistaken.—Mistaken for Disease of the Spinal Cord or Uterus.—Locomotion is Difficult or Impossible.—Patients suffering from it have a Peculiar Walk.—Pain in Pelvic Symphyses.—Constriction by a Bandage at once facilitates Walking.—Conditions to be fulfilled by the Bandage.—Puerperal State may lead to Suppuration of the Pelvic Articulations and Death.

GENTLEMEN :—I have pointed out to you the errors in diagnosis and treatment which displacement of the kidney may occasion.

To-day, I propose to speak to you of an affection which is, generally, not one of gravity ; but which, nevertheless, may embitter a woman's life, by leading her to suppose that she has a disease of the spinal cord or uterus, when there only exists a loosening of the pelvic symphyses.

Before recalling to your recollection the two cases which you have had an opportunity of observing in the clinical wards, I will describe the cases of the two ladies in whom I first observed this affection, which till then had escaped my notice.

Madame X. was married at the age of twenty-three. Her first child was born a year after marriage; a second was born two years later; and a third, when she was more than thirty-six years of age. Her husband had not always been a well-conducted man. Four or five years after his marriage, I treated him for constitutional syphilis. Soon afterwards, his wife had exostoses and baldness, symptoms which led me to recognise in her the same malady for which I was treating her husband. These occurrences took place long before the lady's last confinement. The child was born without any sign of syphilis; and still enjoys good health. Labour was natural, and all went on favorably after delivery. There was nothing unusual in the size of the child's head. From excess of precaution, I desired this lady to keep her bed for fifteen days, and to lie on the sofa for another similar period. I then allowed her to get up. When she tried to walk in her bed-room, she complained of pain in the kidneys and throughout the pelvis: more than a month elapsed before she was able to walk round her room. As she had a little leucorrhœa, I thought slight metritis, a rather frequent sequel of delivery, might be the cause of all her symptoms. I recommended injections, and waited. But one day, when visiting her, I saw her rise from her seat to walk, and was struck by her manner of proceeding: she waddled, dragging one leg after the other with difficulty, leaning greatly to right or left according to the foot she was advancing. She could not stand on one foot; and, on attempting to do so, became doubled up, complaining of acute pain in the hips and loins. It appeared that what she complained of was a feeling of extreme weakness. The possibility of a spinal affection flashed through my mind; but on carefully investigating her sensibility and movements, I ascertained that the skin had lost none of its tactile aptitudes, and that the different movements were well performed, provided the horizontal position was maintained. It then occurred to me, that there might be separation of the symphyses. As the patient was very plump, it was impossible for me to make out the existence of this separation, as I so easily did in the woman who lay in bed 13, Saint-Bernard's ward, whom you all examined.

Pain, somewhat acute, was produced, however, on pressing the mons veneris at the symphysis of the pubes, and on pressing the hips in the situation of the two sacro-iliac symphyses. I forthwith rolled a folded sheet round the hips and pelvis, drawing it as tightly as possible. I then told the patient to walk, which she did at once with the greatest ease, astonished, moreover, to find that her strength, which she had considered as lost, was restored, and that the pains were gone. I directed her to make a laced bandage of drill, so constructed as to encircle tightly the whole of the pelvis and upper part of the thighs. From the time that she began to wear this apparatus, she was able to go about her household affairs, and take some walking exercise. Six weeks later, she was able to discontinue the bandage. Her recovery was complete.

Some time after the occurrence of the case I have now related, a lady was brought to my consulting-room: she was twenty-five years of age, the wife of an officer of one of our special schools. Her husband had carried her to the top of my stair, yet it was with great difficulty that she walked to the sofa. She had not come to consult me for this supposed weakness of the legs, which she considered as a sequel of delivery prior to menstruating. She came to be cured of a very painful temporo-facial neuralgia, for which she had already tried a multiplicity of means of treatment. My prescription proved very useful to her. A month later, she asked me to call on her. It was on that occasion, after thanking me for having cured her neuralgia, that she spoke to me of an affection which, on the first occasion, had very little attracted her notice. She told me that she had had two confinements, quickly following one another, unattended by any untoward occurrences. Her last confinement had taken place three months previously, leaving what she believed, was debility of the legs preventing her from walking. She had no uterine lesion. Leaning on two arms, she walked with great distress, dragging her feet like a paralysed person: when she attempted to raise the leg, as one does to step forward, the other leg, having then to sustain the entire weight of the body, immediately bent under her, causing her to fall, unless supported by some one. She was quite unable to walk alone. I ascertained, just as I did in the case of the other lady, that there was no paralysis: and it at once occurred to me, that I might succeed in this, as I had in the former case. I tightly applied a small table cloth [*naperon*] round the pelvis, and upper part of the thighs: as soon as this impromptu bandage was fixed, I asked the lady to lean on my arm, and try to walk; which she did, but, at first, very timidly. Feeling no pain, by degrees, she gained confidence, and leaned less and less on my arm. When she had reached the end of the drawing-room, I asked her to walk back alone, which she did with a good grace, and with great joy. I then had made for her a doe-skin girdle, so formed as to encircle tightly the pelvis and both trochanters. A fortnight or three weeks later, I had the satisfaction of again seeing this lady in my consulting-room. She continued to wear the bandage. She had ascended my stair without difficulty. She told me, that she had been trying to walk out a little. Two months later, her recovery was complete.

You recollect the big wench who occupied bed 20 in the nursery ward. She was brought to the hospital some days after her second confinement. There were neither uterine nor peritoneal symptoms to make us uneasy. As she was in a perfectly satisfactory state of health, I was not at all thinking about her, when the nurse told me that this woman was quite unable to stand on her legs when she rose in the morning to make her bed. I examined her with care. I ascertained that she had no lesion of the uterus, and that when in bed, she could pretty easily perform all movements. Sensibility was unimpaired. However, even when in bed this woman felt pains in the sacro-iliac and pubic symphyses. It was rather distressing debility than real pain [*une faiblesse pénible qu'une véritable douleur*] that the patient experienced when she moved. She had difficulty in standing, unless she steadied herself by her hands; walking taxed her utmost powers, obliging her to drag her legs, to stop every three or four steps, and to lean on some one's arm or on the back of a chair. She walked from bed to bed, laying hold of the iron bars of the beds. Pressure on the pelvic symphyses did not occasion pain: there was neither swelling nor redness in that situation. There was only a little loosening of the articulations, a relaxation of which the patient was conscious, and of which the physician became aware when he tried to move separately the iliac bones. I pointed out to those who followed my hospital visit, that it was possible to restore the use of her legs to this woman by constriction with a bandage. I at once made the experiment. A bandage was applied very tightly round the pelvis, so as to render the articular surfaces immoveable; immediately, the patient was able to walk. She only remained at the Hôtel-Dieu till a suitable drill bandage was made for her use.

I have learned from persons who have seen this woman, that she has quite recovered. She is able to go about her rather hard work as a charwoman, without fatigue or discomfort.

On the 12th July last, a tall, well-made, robust woman twenty-four years of age, was admitted to Saint-Bernard's ward. She had been confined, for the first time, on the 19th of June. The labour was in all respects good, except that it was somewhat painful from the great size of the child. On the ninth day after delivery, she wished to get up; but found it absolutely impossible. From the time she made the attempt, she experienced great debility in the inferior extremities, as well as an acute pain in the genital organs, a pain which she compared to the sensation of an existing obstacle.[1] She likewise experienced a feeling of weight in the loins.

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[1] "douleur qu'elle comparait à la sensation d'une "*barre.*" The word "*barre,*" in obstetrics, signifies a projection or prolongation of the symphysis pubis; and a female who has this pelvic deformity—a cause of difficult labour—is termed "*une barrée.*"—Translator.

From that time, she suffered the same distressing sensations when she attempted to turn quickly in bed, as when she stood or tried to walk.

Consequent upon this first unsuccessful attempt to get up, this woman took to her bed for some days longer, hoping that more prolonged rest might restore her strength and free her from pain. This result was not obtained. When she renewed her attempt to get up, she found it as impossible to walk as on the former occasion. It was then—the twenty-third day after delivery—that she resolved to come into the hospital.

On her admission, I ascertained, that standing gave her very great pain, and that walking occasioned acute suffering. It was observed, that she immediately edged herself backwards and turned over on her bed, in which she begged to be at once replaced. When interrogated as to the nature and seat of her sufferings, she complained of the genital organs [*des parties génitales*], without more precisely indicating the specially painful parts.

As the general state of the patient was very good, as she had a satisfactory appetite, and was free from fever, it was improbable that pains so acute could depend on inflammation of the uterus or its annexes: my attention was, therefore, at once directed to the state of the pelvic symphyses. In exploring the hypogastrium, I was able to demonstrate to you, that in place of suffering pain in the "genital organs," as she stated, the woman was suffering in the pubic arch, the sole seat of pain being a point corresponding to the pubic symphysis. By palpation in that situation, a very considerable separation of the articular surfaces was recognised: the extremity of the index finger could easily be introduced between the two bones of the pubes: and it could, at the same time, be felt, that the interarticular cartilage was softened. This exploration was very painful. I avoided trying to move the one bone on the other: this experiment, which would have caused great pain, would not have given me any additional information: it was clearly a case of loosening of the symphysis pubis. To make certainty more certain, I examined the internal genital organs, and found that they were in a perfectly healthy state.

When in bed, this woman could move her legs perfectly well; and she made no complaint of any symptom of paralysis. There could, therefore, no longer be any doubt as to the nature of the case: the symptoms were evidently dependent on disjunction of the symphysis pubis.

Had there still remained the possibility of doubt in the diagnosis, it would at once have been dispelled by the result of the treatment. I girded an abdominal bandage on the hips, pressing tightly round the pelvis, including trochanters and pubic arch. No sooner had this improvised bandage been applied, than the woman, who a minute before could not stand, was able to walk easily, carrying her infant in her arms at the same time.

I had all the trouble in the world to get her to remain some days in the hospital. She wished at once to leave on foot. She only remained till her bandage was made. I had it constructed with the greatest possible simplicity—that is to say, it was a strong broad girdle, wherewith to encircle the sacro-iliac symphyses, the trochanters, and the pubes.

You will remark, Gentlemen, how easily a superficial observer might have formed an erroneous diagnosis in this case. The woman had been recently delivered: she vaguely complained of pain in the genital organs: she said she could not walk without pain: in point of fact, she could not walk. Would it not have been very natural, in the first instance, to conclude, that the patient was suffering from a lesion of the uterus? But this error could not have been committed, if, in interrogating the patient, she had been pressed to indicate the precise point where she felt pain, the finger being forthwith employed to make such an examination as you saw me make: there would then have been ascertained—what you saw me ascertain—separation of the bones, and softening of the cartilages.

Errors in diagnosis are apt to arise from the existence of lumbar and hypogastric pains. There may be leucorrhœa: on digital examination, we may discover lacerations or persistant granulations of the cervix uteri: with such symptoms existing, and no additional data except the vague statements of the patients to guide us, the impossibility to walk or stand might quite naturally be attributed to metritis. The mistake is the more excusable, that metritis occurs very often, and loosening of the symphyses very seldom.

I have no claim, Gentlemen, to having made a discovery. Disjunction of the pelvic symphyses has been pointed out by obstetricians. They have even tried to explain it by the great size of the head of the fœtus: they have suggested, that by acting like a wedge, it separates the previously softened symphyses. This probably occurred in the case I last related to you. The woman said that her infant was very large.

In this lecture, I have been particularly anxious to call your attention to an affection of rare occurrence, and consequently little known. So little is it known, that some of your text-books on midwifery, including Cazeaux's treatise, do not even mention it as a possible sequel of labour. There is an indisposition to admit the presence of a pathological state of rare occurrence—a tendency consequently, to attribute symptoms rather to an anomalous metritis than to a loosening of the pelvic symphyses. I wish you to be on your guard against committing a mistake of this kind.

Loosening of the pelvic symphyses is so far a serious affection that it absolutely prevents walking, the inability increasing, the longer the patient makes no attempt to walk. You saw that the patient whose case I first related to you could with difficulty walk a few steps in her apartment, two months after delivery: that the second patient, three months after delivery, could not walk at all. There is no physiological reason why such a condition should not continue indefinitely. We have learned from experience, that rest alone is not sufficient to cure the diastasis when it is considerable. To promote consolidation, it seems necessary to bring the articular surfaces into contact with one another by artificial means.

From these considerations, you can at once deduce the proper treatment. As you know, man, being a biped, requires a pelvis sufficiently solid to be proof against all trials of strength in walking. If the sacrum could move on the ischium, or if the bones of the pubes were not solidly united, walking would be impossible; for the weight of the body would then inevitably cause disjunction of the bones of the pelvis. When loosening of the pelvic symphyses is an obstacle to locomotion, they must be artificially consolidated. A girdle requires to be placed round a pelvis which has its staves separated: it is necessary to supply the temporary deficiency of intrinsic contention by an extrinsic contention—that is to say, by tight application of a bandage, in such a way as to bring into contact the separated surfaces of the symphyses.

As you have seen, this bandage can be improvised. A strong towel properly tightened answers quite well. But to secure a solid durable apparatus, it is better to have a bandage made of strong drill or doe-skin, which can be laced more or less tightly at pleasure; and which is constructed in such a way as to embrace the trochanter, as well as the bones of the pelvis. Should such an apparatus be found insufficient, a steel spring may be added to it, arranged so as to make simultaneous pressure on the sacrum, ilium, and pubes. If the loosening be considerable, and the pain very acute, rest must be enjoined: but let me repeat, that rest is not enough by itself: to wait for consolidation would be losing time: it is absolutely necessary for the patient to have recourse to an apparatus till she can walk without its aid.

We have seen how a bandage of the simplest possible description may relieve and rapidly cure loosening of the symphyses. I cannot terminate this lecture without endeavouring to explain to you how this pathological softening is merely the exaggeration of a physiological state, having as its final cause the facilitating of the exit of the fœtus. During pregnancy, the pelvic articulations gradually lose, to a certain extent, their intimate union: the ligaments become relaxed, permitting during labour, a minute augmentation of the diameters of the pelvis to allow the head of the fœtus to traverse the outlet more easily. This physiological loosening may become excessive, and render walking difficult during the later weeks of pregnancy: and walking may even be rendered completely impossible by the wide separation of the pelvic symphyses which has taken place during labour.

I have said enough on this point. Let me now remark, that in consequence of the existence of the puerperal state, inflammation may complicate *post partum* loosening of the pelvic symphyses, and lead to death.

A woman, forty years of age, occupying bed 3 of Saint-Bernard's ward, was admitted to our wards some weeks after her confinement, in consequence of a pain in the right iliac fossa, and the continuance of fever from the date of her delivery. She had become very thin, and was losing strength day by day. She had nearly altogether lost her appetite: my *chef de clinique* observed that every evening she became very hot, that condition being generally preceded by rigors. The progress of the disease, and the absence of characteristic symptoms, afforded no ground for concluding that the case was one of purulent infection or continued fever. There was no pulmonary affection to explain the paroxysms of fever. The uterus was not the seat of pain: there was no discharge: there was no pelvic abscess:

and the right iliac fossa, the seat of pain, did not contain a tumor. One day, the patient told me that the pain had extended to the right hip. My first examination was without result: but at an interval of some days, as the pain continued, I more carefully explored the region of the nates, and found that there was œdema in that situation, that pressure there occasioned pain, particularly over the right sacro-iliac symphyses. I made an exploratory puncture with a capillary trocar, by which I withdrew several drops of greenish fœtid pus. The patient left the hospital; so that the history of her case is incomplete.

Some months later, in October 1862, a patient was admitted to bed 30 Saint-Bernard's ward, four weeks after her confinement. Five days after delivery, she had felt pain in the right hip of so acute a character as to prevent her from getting up. Some days later, she was seized with rigors and fever; and for three weeks prior to admission, she had never been without fever. The pain had invaded the right hip, and the symphysis of the pubes. The right hip was painful; and although the patient presented none of the symptoms of purulent infection, I had no hesitation in saying that in all probability the sacro-iliac and pubic symphyses were the seat of an inflammation which explained the constant fever. The frequent rigors, I regarded as indicating suppuration of the weakened joints. Both hips soon became œdematous, and gave clear evidence of deep-seated fluctuation. I introduced a bistoury in the direction of the right and left sacro-iliac symphyses. A great quantity of pus flowed from the incision: the opening was prevented from closing by the introduction of a dossil of charpie. I had formed an exceedingly unfavourable prognosis. The fever continued: and the patient died a few days after the opening of the abscess.

At the autopsy, it was found, that the sacro-iliac symphyses were denuded of their cartilages; and that the greater part of the articular surface showed the appearances of ostalgitis. There was pus also in the symphyses of the pubes, but it was in small quantity; and in some places, only the cartilage was frayed. No morbid appearances were presented by the uterus or its annexes. No metastatic abscesses were found in the lungs, liver, or any other organ. In the right iliac fossa, under the muscle, there was an abscess communicating with the corresponding articulation.

Bear in mind, Gentlemen, that this woman was delivered at her own home, away from epidemic influence. Also, bear in mind, that fifteen days prior to delivery, she had had pain in the right hip. What information are we to deduce from these facts? Are we to infer, that in recently delivered women, loosening of the pelvic symphyses may become so painful as to be accompanied by inflammation, leading sometimes to the most serious consequences? You cannot pay too much attention to pains complained of in the pelvic symphyses: you must place the patients in conditions the most favorable for calming the pain, and arresting the progress of the inflammation, of which pain is sometimes the origin.

LECTURE CI.

PERCUSSION.

Influence of the Sensualistic Philosophy on Contemporary Science and on the Tendencies of the Parisian School.—Pinel, and the Natural History of Diseases.—Pathological and Semeiotic Anatomy inaugurated by Corvisart.—Discovery of Percussion by Avenbrugger, and of Auscultation by Laennec.—Succession of Works on Semciology.—Immediate and Mediate Percussion.—The Plessimeter.—The Plessigraph: manner of using it.—Comparative Value of the Modes of Percussion.—Medicine does not consist solely in the study of Morbid Anatomy and Semeiology.—Micrography and Nihilism in Therapeutics.—Necessity of associating Modern Precision with the Medical Doctrines of Past Times.

Gentlemen:—Before I leave this clinical chair, I wish to speak to you on the subject of percussion; and keeping that object in view, I desire to take with you a rapid glance at contemporary medical doctrines. It may be said, that the dominant characteristic of our medical age is the application of physical methods of investigation, and an ambition to attain in medicine that precision and rigor which belongs to sciences called exact.

You know, Gentlemen, that medicine always takes its inspiration from the reigning philosophy; which means that every philosophical doctrine has, in the history of medicine, a corresponding medical doctrine. The actual medical tendencies are certainly those which the grandsons of the eighteenth century ought to possess. They are, moreover, the universal tendencies of contemporary science.

No one in France, to whatever philosophical religion he may belong, will deny that sensualistic philosophy has exercised a great influence on the scientific progress of our age.

Whether it be to blame or to laud them, it must be admitted, that the philosophers of the eighteenth century, who with one exception, invoke the "*sage* Locke," and derive their inspiration from the sensualism of Condillac, have not uselessly explored French philosophy to its lowest depths.

At the voice of these philosophers, who were by turns scientific, ironical, and impassioned—but always accessible from the simplicity of their language—the era of revolutions opened. On so deeply disturbed a soil, new institutions alone were possible. Social and scientific rights had alike undergone a change.

Medicine came under the same influences; and also had its own revolution. By one of these providential occurrences, of which history presents examples, the needed man—Laennec—appeared: a great discovery was made—auscultation. From that time, the spirit of medical inquiry, obedient to the impulse which it had received, unresistingly surrendered itself to the pursuit of analytical methods.

Laennec's discovery, however, was not an isolated fact: it was the progeny, so to speak, of other discoveries, but its wide-spread celebrity and admirable results gave it a dominant power over medicine, and definitively stamped the movement with a character which it still retains.

Medicine, as I remarked, aspired to precision and rigorous exactness; and wished to be one of the "exact sciences." The study of lesions—that is pathological anatomy—and the study of symptoms—that is semeiology—being the departments of medical science most accessible, even to mediocre intellects, and being departments easily admitting of culture by rigorously scientific means, were at once impetuously taken up. On the other hand, therapeutics, the study of which is infinitely more complex and difficult, became almost entirely neglected. Medicine was thus cultivated for the sake of science, and without a thought—so to speak—as to the means of assuaging the sufferings or promoting the well-being of patients. A disease became a subject of abstract study: it was studied as if it were a plant, an animal, or a physical phenomenon. Pathology became no more than the natural history of diseases.

It was under the influence of that bias that Pinel wrote his Nosography. In systematising morbid conditions, in seeking to classify them according to types representing general species and varieties; he did what was of great use for the cause of study, because the mind likes to repose on a distinctly defined type: but he did an injury to practical medicine, and in the end to science; because he believed, and led others to believe, in the abstract types which he had created.

I say that Pinel did an injury to practical medicine: the young practitioner imbued with his doctrines, when at the bedside of his patient, sought in vain for the well depicted entity of disease which he became acquainted with when a student on the benches of the school. The doctrines to which I refer, led practitioners to believe in the necessary and almost methodical progress of diseases, which naturally produced the serious evil of abstinence from all treatment—of "expectation" in therapeutics.

I say that, in the first place, Pinel inflicted a direct injury on science, because he taught the untruth, that diseases are entities admitting of classification like animals and plants; and, in the second place, he did mischief indirectly to science, by inculcating minute analysis, a searching after the slightest differences in symptoms, which tends to introduce confusion, by infinitely multiplying morbid species and varieties.

Nearly at the same epoch, Corvisart, a man clear in his conceptions, ready and reliable in judgment, lucid and incisive as a speaker, inaugurated the new era of pathological anatomy in France, by passionately espousing the science illustrated by Morgagni. Around him were grouped Dupuytren, Bayle, and Laennec, men whose names were destined to become renowned.

Then it was, that a series of discoveries were made, the filiation of which I now wish to elucidate to you. In what I am going to say, you will find additional proof of the correctness of the statement I have often made to you, that nothing new is ever achieved in science at one single bound. The appreciation of one fact leads to the appreciation of another, as a consequence or corollary from the first: and one discovery begets another discovery. Thus it was, that by a chain of facts and ideas Avenbrugger was the precursor of Laennec.

In 1761, Avenbrugger, a laborious physician of Vienna, published a modest duodecimo entitled:—"*Inventum Novum ex Percussione Thoracis ut signo abstrusos interni pectoris morbos detegendi.*" This title had the double draw-back of being too long, and of not being very intelligible. Avenbrugger's discovery, the starting point as you will see of all modern works on precise observation, made no impression in Germany. The author had timidly and indirectly, but uselessly, placed his work under the patronage of "the very illustrious Baron Van Swieten:" the book had but little success: the inventor of percussion lived in obscurity, and died unknown. In 1773, Rozière de la Chassagne translated Avenbrugger's work into French.[1] The translation had as little success as the original. Corvisart, imbued with the positive doctrines of his time, and searching for the means of discovering during life the lesions found after death, having seen in Stoll, his favorite author, that information such as he desired could be obtained by percussion of the chest in cases of disease of the thoracic organs, resolved to read the entirely forgotten work of the German physician. This resolution brought good fortune to Avenbrugger's discovery, though not perhaps to his book.[2] Corvisart immediately made experiments in respect of percussion; and for twenty years, he practised it before his clinical pupils at the Hôpital de la Charité. Nor was that all: when palpation and percussion failed to furnish him with sufficient data, he was in the habit of applying his ear to the chest of the patient to enable him better to distinguish the sounds of the heart. Thus, Corvisart supplemented insufficiency of touch by the aid of hearing. You perceive, how one method of physical examination thus led to the discovery of another physical method. But though Corvisart was possessed of a talent for diffusing knowledge, he was not equally endowed with a talent for invention. By a small intellectual effort, he might have rendered his name immortal by the discovery of auscultation. This effort he failed to make. Among his pupils, however, there was a young physician, a man of grave and meditative mind, full of ancient lore, and yet inspired with the spirit of modern inquiry. Laennec, following his master's example, applied his ear to the chest; but he was more fortunate than his master in knowing how to listen and understand. He discovered auscultation![3]

Laennec has himself related how he came to make the discovery—how he came to think of practising mediate auscultation. In 1816, half a century after the discovery had been made by Avenbrugger, he was consulted by a young woman who had the general symptoms of heart-disease, and in whom, from her plump condition, the application of the hand, and percussion, afforded little insight into the nature of the malady. Laennec, "on account of the age and sex of the patient," felt himself interdicted from applying his ear to the chest: he recollected "a very well-known acoustic phenomenon, viz. that if the ear be applied to one extremity of a post, the stroke of a pin at its other extremity will be heard very distinctly." He then applied to the precordial region one end of a very tightly compacted roll of paper, and placed his ear at the other end of it. He "was equally surprised and pleased, to hear the pulsations of the heart with much more precision and distinctness than by the immediate application of the ear." It was not by chance, therefore, but by reasoning, that Laennec discovered auscultation: in the true meaning of the word, he *invented* auscultation, because he *sought* for it, the means of search he employed being induction.

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[1] As an appendix to his "Manuel des Pulmoniques:" Paris, 1773.

[2] "La Nouvelle Methode [d'Avenbrugger] pour connaître les Maladies Internes de la Poitrine par la Percussion de cette Cavité: traduite en français par J. N. Corvisart:" 1808.

[3] See LAENNEC's work entitled: "De l'auscultation Médiate: ou Traité du Diagnostic des Maladies des Poumons et du Cœur."

The physical examination of the chest, installed with so much eclat by Laennec, suggested the examination of, if not all the organs, at least the products of secretion: the urine was submitted to the action of chemical reagents, as was likewise the blood, the great nutrient secretion. Albuminuria was discovered: and glycosuria became better known. To the anæmia of the old physicians, were added uræmia, leucocythæmia, and melanæmia. Examination by the speculum was brought to perfection and generalised—too much generalised perhaps: the laryngoscope was used for examining the larynx: the ophthalmoscope was used for examining the eye—the interior of the eye—that expansion of the brain—by ascertaining the state of which we can sometimes judge of the state of the encephalon. The most inaccessible organs can now be seen. These conquests of modern semeiology are due to the spirit of investigation developed in the contemporary generation by the successive discoveries of Avenbrugger and Laennec.[1]

Let me now return to the subject of percussion. Avenbrugger recommended percussion to be performed by striking the chest slowly and gently with the extremities of the extended fingers placed close to one another:—"*Percuti, verius pulsari thorax debet, adductis ad se mutuo et in rectum protensis digitorum apicibus, lente et leniter.*"

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[1] See the Lecture on APHONIA AND CAUTERISATION OF THE LARYNX: Volume III., p. 105, and the Lecture on CEREBRAL FEVER, Volume I., p. 451.

To this method of employing percussion, Corvisart added percussion with the open hand [*percussion à main ouverte*], which, he said, is "a method exceedingly useful for ascertaining the extent of the part of the thorax which is not resonant, and appreciating more correctly the amount of the obstacle."

Avenbrugger's method is suited for ascertaining differences in elasticity, that is to say, differences in the consistence of the parts percussed: but, strange to say, he never mentioned this, and very probably was not aware of it. He speaks only of the difference of sound. However, one obtains a much better knowledge of difference of consistence by striking with the pulp of the extended fingers than with the open hand as Corvisart recommended.

It is also very remarkable, that *Avenbrugger really practised mediate percussion:* he recommended percussion to be made over the shirt rendered very tense, or with a gloved hand, provided the glove was not made of smooth leather:—"*Thoraci supertensum sit indusium vel manûs percutientis chirothecâ* (*modo ex polite corio non sit*) *muniatur.*" Corvisart regards this precept as useless, believing that it matters not which way is adopted. Avenbrugger was careful to add a note, stating that in percussion of the naked chest by the naked hand, the contact of the smooth surfaces produces a particular sound (*strepitus*) which modifies the quality of the real sound which ought to be elicited:—"*Si nudum pectus nudâ manu pulsatur, superficierum politarum concursus strepitum producit, et soni evocandi constitutionem obscurat.*" It is very evident that Avenbrugger wished to get rid of the noise of the skin, or at least to deaden it, so as to get the pure sound from the deep-lying parts; and that he practised a species of mediate percussion. In place of employing a plate according to the modern plan, he interposed a piece of stuff or leather between his fingers and the chest; and in place of striking with the open hand, he percussed with the extremities of the fingers, exactly as has been recommended, long after his day, by the advocates of mediate percussion.

We have seen that Avenbrugger percussed with an intermediate piece of stuff or rough leather; and Corvisart regarded the use of an intermediate substance as a matter of indifference. There is some uncertainty as to who it was who first employed the finger as the medium in percussion: but it was the mode of percussion habitually adopted by Récamier. Dr. Piorry proposed to substitute for the interposed finger, a plate, which he called a *plessimeter*. The innovation was a real advance. You are all acquainted with Piorry's instrument, which is a plate with ears at the sides to enable it to be firmly held in the left hand. It is sometimes made of metal; but the plessimeters most used are of ivory. One of my pupils, M. Horteloup, son of my good friend and colleague of the Hôtel-Dieu, has had a plessimeter made of gutta percha, which has not the disadvantage of giving the dry sound of the ivory. Another of my pupils, M. Oldfield, has brought out a percussion hammer, formed of a flexible whalebone stem terminating at one of its extremities in a very hard sphere formed of plates of leather very tightly compacted. Thus, we have a hammer and plessimeter formed of two substances nearly identical, which do not vibrate in the least degree, and yield with great purity the sound of the percussed parts. These are the two instruments which you generally see me use. I find them very serviceable; and, contrary to what may be supposed, I obtain by their aid a very precise conception of the consistence of bodies.

In Germany, very much use is made of a plessimeter half the size of that employed in France, and of a small steel hammer, or metallic hammer covered with caoutchouc.

The plessimeter, so long as it is not childishly used as a toy in cases where it is not required—so long as it is not employed to percuss a bone or an artery, the limits of which can be quite well determined by the eye and palpation—so long as it is not made a pretext for plessimetric exercises under which patients' common sense, and truth equally groan—the plessimeter is a good instrument, an instrument which renders essential service.

Percussion on the finger or plessimeter has the very great drawback of not giving a *simple*, but a *mixed* sound. At the junction of organs, the finger, and still more the plessimeter, are over two or more organs, which percussion causes to vibrate simultaneously, so that the sound elicited is a mixture of two or more orders of vibrations. For example, at the point where the liver and right lung intersect, the finger or plessimeter is generally placed over the liver and lung, in such a way as to give a mixed sound on percussion, which is neither the sound of the liver nor of the lung. The sound is much duller than that of the lung, but it has not the dulness which would be obtained were the liver isolated when percussed. To ascertain the exact point at which the liver commences, it is necessary to feel one's way, to percuss a little above and a little below the place where the mixed sound begins, and after thus groping, the operator, if aided by great practice, discovers with considerable precision the point at which the liver begins. Similar remarks are applicable to percussion of the heart at the mediastinum. Again, we generally lose trace of the heart at its left margin, where it dips down into the chest, hiding itself, as it were, behind the lung.

The ideal of percussion, then, is to percuss the smallest possible surface, so as to disturb the smallest possible portion of an organ—to percuss in such a way, that at the distance of a few millimeters, the space not percussed should not be made to vibrate, and should not mingle its sounds with the sound of the percussed space. It is then very evident, that immediately the sound changes, we are within a few millimeters of the limits of another organ. To attain this ideal, Dr. Michel Peter, my former *chef de clinique*, devised an instrument by which he reduced to a minimum the percussed surface, by reducing to a minimum the percussing surface. As a percussing surface so exceedingly reduced could yield very little sound, the idea occurred to him of amplifying the sound by a supporting stem. In other words, he devised an instrument for percussion consisting of a cylindrical stem, the percussing extremity of which terminates in a truncated cone, and the percussed extremity in a disc larger than the cylindrical portion, and on the flat surface of which the percussing finger easily strikes. The circular plane which rests on the skin does not contain more than five or six square millimeters: it necessarily does not vibrate, nor cause to vibrate a greater extent of surface. In percussing, the instrument is moved progressively in a line, every point in which is tested, so that as soon as a change in the nature of the sound is heard, we know within about one or two millimeters—and without any groping [*sans aucun tâtonnement*]—that a new organ has presented itself under the instrument of percussion.

Dr. Peter's instrument is provided with a crayon, or (which is more simple) a slightly carbonised cylinder of cork, which moves with gentle rubbing, so that by pushing it out, a black mark can be made at every point where there is a change in the nature of the sound. The instrument, therefore, is at once both an agent for percussion and delineation. You can understand how greatly its use will shorten an investigation, by rendering it both more convenient and more precise: it obviates the necessity of holding in the hand which percusses, the pencil with which the limits of organs are marked.

Nor is this all: this instrument of Dr. Peter—which he calls a plessigraph—is exceedingly delicate. It is not necessary to *percuss* —it is sufficient merely to *touch.* The touch ought moreover to be as rapid as possible.

Let me now describe the manner of using this instrument:—Hold the instrument firmly between the thumb and index finger of the left hand: the less you put the fingers on it, the less you deaden the sound. Place the thumb on the little knob by which the crayon is moved. Holding the plessigraph, as I have now described, in the left hand, apply it firmly and perpendicularly to the surface of the skin.

Touch the firmly held plessigraph with the palmar surface of the extended index finger of the right hand. Do not *percuss,* as when you use the plessimeter, with the ungual portion of the fingers flexed. You thus avoid hard striking, which might be painful from the smallness of the surface percussed: as the plessigraph is touched by the whole palmar surface of the index finger, and not by the ungual extremity, an incredible delicacy of sensation is obtained. The consistence of tissues is perceived with very great precision, which you can understand, as they are touched by the intermediary of the plessigraph just as in performing palpation for the discovery of the nature of bodies.

Dr. Peter's instrument is ten centimeters in length: it is divided into ten equal parts, each of which is one centimeter: one of these centimeters is divided into millimeters; so that the instrument, besides serving for the percussion and delineation of organs, serves also for their measurement.

To sum up:—The plessigraph appears to me fitted to measure organs with extreme precision and with great rapidity. There is no difficulty in using it: the most inexperienced pupil can easily employ it, and obtain, on the first occasion of employing it, results which are not attainable with the plessimeter till after long experience and protracted groping. It has the additional advantage of leaving a series of black points wherever a difference of sound has been detected: whenever the limits of an organ have been ascertained, they become likewise delineated, so that the operator *sees* that which he has *heard:* the image remains permanently under the eye, long after the sound has ceased. You remember the precept of Horace :—

> "Segnius irritant animos demissa per aurem :
> Quam quæ sunt oculis subjecta fidelibus."

Once more in conclusion :—When one percusses with the extended fingers closely approximated, following Avenbrugger's method—when one percusses with the open hand in accordance with Corvisart's teaching—when one percusses on the finger, on the plessimeter, with or without plessigraph, one does well, provided examination by percussion be indicated. Here, however, a distinction must be made. The immediate percussion of Avenbrugger and Corvisart is preferable for an examination of the chest, with a view to ascertaining at once the general state of the cavity as to sonorousness. It is generally sufficient for the recognition of a pleuritic effusion or a moderately extensive pneumonia. Percussion on the plessimeter is not suited for the examination of the apices of the lungs, particularly for their examination behind. You have seen me sometimes percuss on the clavicle, with the extremity of the fingers using it as a plessimeter; and by this proceeding, I have obtained very satisfactory results in cases of pulmonary tuberculisation. But for investigations in which precision is necessary, when it is desired to ascertain the exact limits of the liver, spleen, and other organs, particularly of the heart, and the exact limits of tumors of the mediastinum, such as a deep-lying aneurism of the aorta, the plessigraph seems to me to be preferable in many cases.

Gentlemen, if after mature reflection, we dispassionately sum up the balance sheet of our knowledge, if, free from the passions which moved our predecessors, we endeavour to assign to every one his just share, if we ask ourselves the value of those instruments of analytical investigation in our hands, if we ask ourselves whether they constitute the whole of medicine, we are forced to answer by an emphatic negative. No: the knowledge they yield does not constitute the whole of medicine.

It verily seems, I deliberately repeat, as if the medical intellect had been upset by Laennec's discovery. Physicians rushed into excesses in physical inquiry: one would give the medical world his *petit bruit de souffle* and another would point out some *nuance* which had been neglected by the otherwise comprehensive genius of Laennec.

There also arose the strange idea that, because henceforth lesions and their limits were more ascertainable, the means of curing them were better known. Medicine, it was supposed, was about to rival surgery in precision. This, however, was an enormous mistake; for although surgery sees clear as day the lesions confided to it for cure, it certainly cannot on that account cure them any better.

Assuredly, Gentlemen, we owe much to our predecessors and contemporaries: they have advanced diagnosis to a marvellous degree of precision; but in so doing, they have only advanced semeiology. Medical science has progressed; but the art of healing has remained nearly stationary. In therapeutics, experiment is much more difficult: the data of the therapeutic problem are so numerous, the results so uncertain and deceptive, that it is impossible to arrive at a conclusion rapidly; and the conclusion, when attained, is far from being always susceptible of rigorous demonstration.

It is still an open question whether micrography in its most advanced development—the *cellular pathology* of Virchow—by reviving under a new scientific form more suited to our age, the Epicurean system of atoms, does not lead directly to the annihilation of therapeutics. By regarding the living organism as a microcosm formed of heterogeneous and independent elements, it necessarily rejects all general medication, which can produce no influence on elements which are incongruous, and, to a certain extent, antagonistic. It forgets the man in thinking of the cell; and loses itself in an abyss of infinitesimals.

When medicine as it now exists compares what it knows with what it does, it perceives that pathological anatomy does not always necessarily lead to rational therapeutics; and that the knowledge of lesions does not always enable us to cure them. Here, the deception begins. Too much being hoped for, disappointment comes too quickly: the descent from disappointment to scepticism is very rapid. Gird up yourselves, young men, to resist such tendencies! Rich in the possessions bequeathed to you by the physicians of the past, to you belongs the duty of uniting modern science with ancient wisdom, and of rekindling the temporarily despised torch of old medical traditions. Let this be your endeavour! It is a great and splendid aim! Fail not to pursue it!

With this exhortation I conclude.

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